SURGERY

VOL. 48 AUGUST, 1960 No. 2

Original Communications

PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAPHY IN THE DIAGNOSIS OF POSTHEPATIC JAUNDICE

MANUEL SANTOS, M.D., LUIS FIGUEROA, M.D., AND OSCAR LÓPEZ, M.D.,
SANTIAGO, CHILE

(From the Department of Surgery, Division B, José Joaquín Aguirre Hospital, University of Chile)

PERCUTANEOUS transhepatic cholangiography is a valuable procedure in determining the origin of jaundice, whether hepatic or posthepatic, especially when other exhaustive investigations have failed to reveal the cause of jaundice.

This procedure is also useful in patients with prolonged extrahepatic jaundice in distinguishing the cause and level of a common bile duct obstruction.

Despite thorough preoperative studies of the patients, Zollinger[36] has reported a 16 per cent error in etiological diagnosis of posthepatic jaundice; 6.4 per cent of his patients with intrahepatic jaundice were subjected to laparatomy. Since the patient with prolonged jaundice is a poor surgical risk, the surgeon faces the responsibility of making a correct diagnosis without laparotomy.

Since in patients with posthepatic jaundice there is dilatation of the hepatic biliary radicles, percutaneous transhepatic cholangiography allows visualization of the biliary tract by injection of dye into a dilated duct within the parenchyma of the liver.

The method of percutaneous transhepatic cholangiography was first described in 1952 by Carter and Saypol.[3] Subsequently, important contributions on visualization of the biliary tract by this method have been made by Garcia Siguero,[9] Halligan,[12] Varela Fuentes,[33] Kidd,[13] Wilks,[34] and many others. Since none of these authors have described in detail the patients' studies or given the indications for the use of percutaneous transhepatic cholangiography, it is difficult to draw firm conclusions from their work.

The patient selected for percutaneous transhepatic cholangiography should be hospitalized. All existing methods for determining whether the jaundice is hepatocellular or posthepatic should be used and analyzed in detail. A prolonged prothrombin time is a contraindication for hepatic puncture, and this

Received for publication Nov. 30, 1959; resubmitted Jan. 18, 1960.

procedure should be postponed until the prothrombin percentage is close to normal. Exception is made in acute cases when biliary decompression is urgent.

METHOD

Preparation.—The evening before examination, enemas are given until the return is clear; a mild sedative is administered. The patient should be taught

Fig. 1.

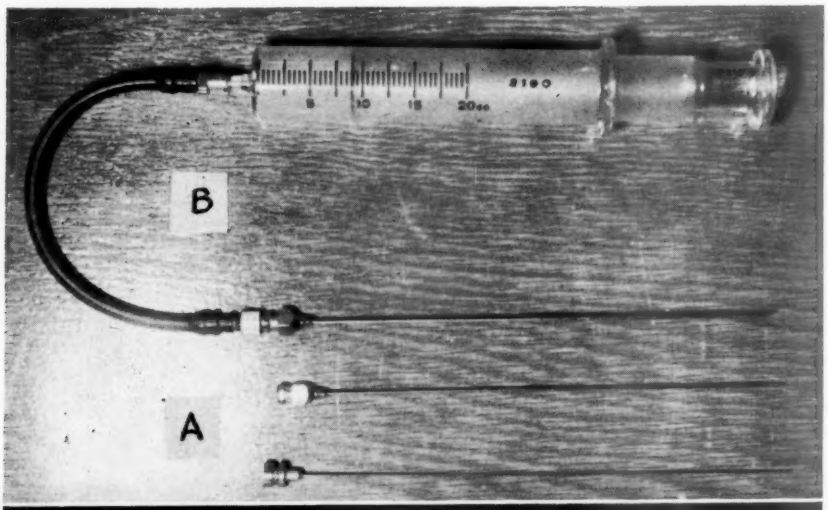


Fig. 2.

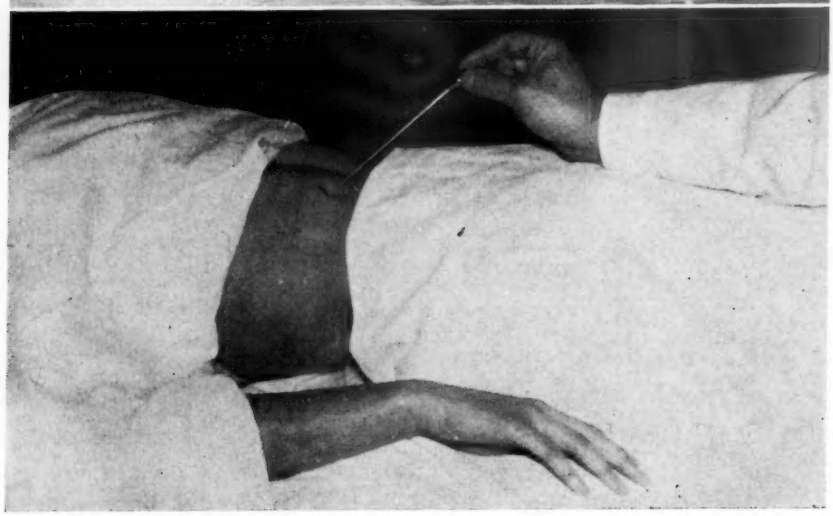


Fig. 1.—Instruments required for percutaneous transhepatic cholangiography: Trocar approximately 12 cm. long and 1.3 mm. in diameter, two 20 c.c. syringes, and a soft rubber connection tube.

Fig. 2.—Position of the patient on the x-ray table, site of puncture, and direction of insertion of the needle.

to hold his breath in expiration for a prolonged period. One hour before percutaneous transhepatic cholangiography is begun, another sedative is given. The liver is carefully palpated and pressure is applied to rule out the possible presence of an intestinal loop between the abdominal wall and the liver.

Position and anesthesia.—The patient is placed flat on his back on the x-ray table and the skin is prepared surgically. The abdominal wall, the peritoneum, and the hepatic capsule are anesthetized with 2 per cent Novocain.

Instruments.—A flexible steel trocar and stilette approximately 12 cm. in length and 1.3 mm. in outside diameter, two 20 c.c. syringes, and a soft rubber connecting tube are required (Fig. 1).

Procedure.—The trocar is introduced through the abdominal wall 3 cm. below the xiphoid process and 3 cm. to the right of the midline, directed toward the right lobe of the liver and making an angle of approximately 45 degrees with the horizontal plane (Fig. 2).

When the trocar has penetrated the abdominal wall, the patient is told to exhale completely and hold his breath. At this point, the full length of the trocar is advanced in the direction described. The sensation transmitted on puncturing the liver is characteristic, easily recognized, and difficult to confuse. The patient is told to continue breathing, as soon as the liver substance has been entered. The trocar, now in the liver, should move with each respiration.

The stilette is removed and a syringe is connected; aspiration is constant until bile is obtained. Blood frequently aspirated is an indication that the position of the needle should be changed. When a bile duct has been entered by the needle, the amount of bile aspirated may vary from several cubic centimeters to about 500 c.c. Sometimes, white bile is obtained, or the bile may be dark or purulent. In obstructive jaundice, the intrahepatic bile is fluid and aspiration can be performed with a small needle without difficulty.

The bile may be utilized for bacteriologic, chemical, and histopathologic examinations.

After bile has been aspirated, a syringe filled with Diodrast,* 35 per cent, is fitted to the needle by means of a rubber tube. The dye is injected slowly under radioscopic observation until the intrahepatic and extrahepatic ducts are filled. It is important that the distal portion of the biliary tract be filled with dye. Generally, 20 c.c. is sufficient, but in some cases as much as 60 c.c. may be necessary. Roentgenograms are taken with the patient in different positions, in order to avoid bone overlay of the extrahepatic biliary tree.

Before the trocar is withdrawn, as much bile and dye as possible should be aspirated to prevent biliary hypertension and the possibility of leakage of bile through the site of puncture, with a consequent peritonitis. The patient should remain hospitalized and under close observation for a 24-hour period. Broad spectrum antibiotics are also indicated.

ANALYSIS OF CASES

At the Joaquin Aguirre Hospital, this examination has been carried out on 54 occasions in 46 patients. These patients ranged in age from 11 to 68 years; 25 were between 50 and 68, 17 between 30 and 49, and only 4 younger than 30 years. Twenty patients were male and 26 were female.

Jaundice had been present in these patients for periods varying from days to 4 years, with an average of 10 months and 15 days.

*Winthrop Laboratories supplied the Diodrast, 35 per cent.

Fig. 3.

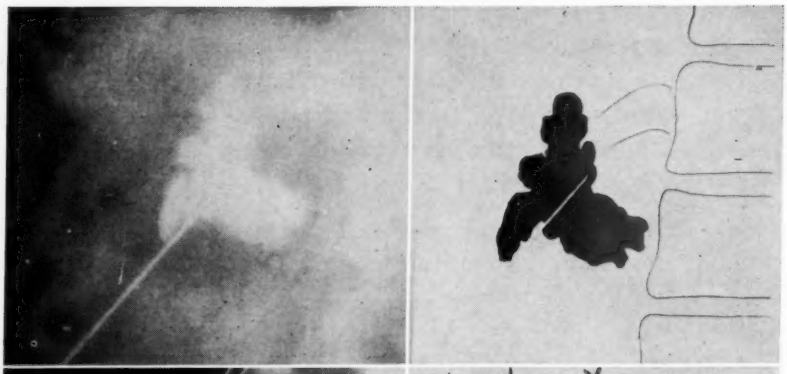


Fig. 4.

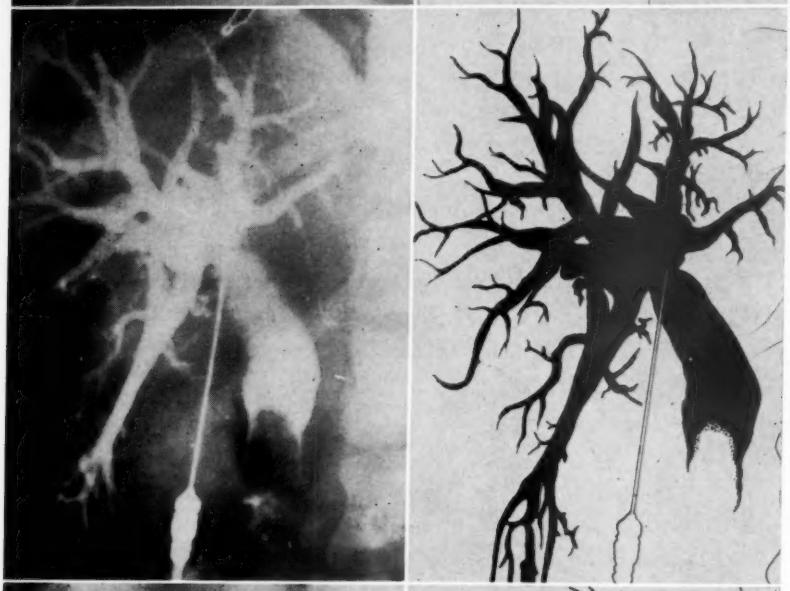


Fig. 5.

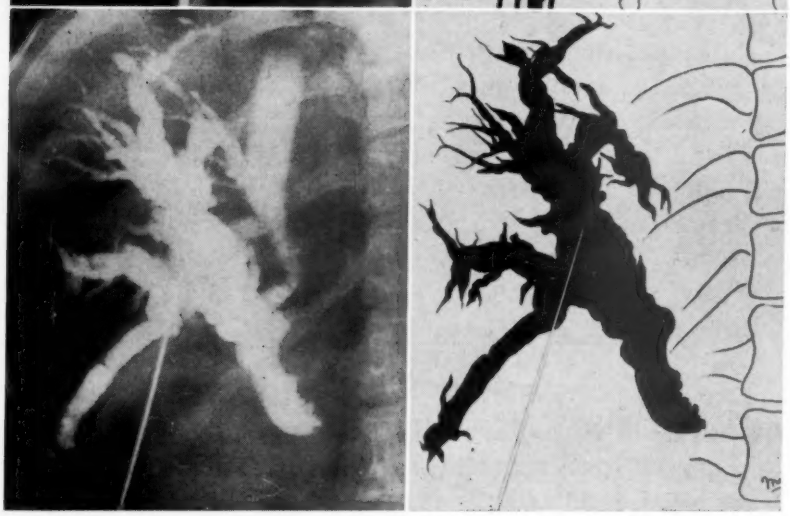


Fig. 3.—Hepatic hydatid cyst.
Fig. 4.—Complete obstructive jaundice due to choledocholithiasis.
Fig. 5.—Complete obstructive jaundice due to cancer of the common bile duct.

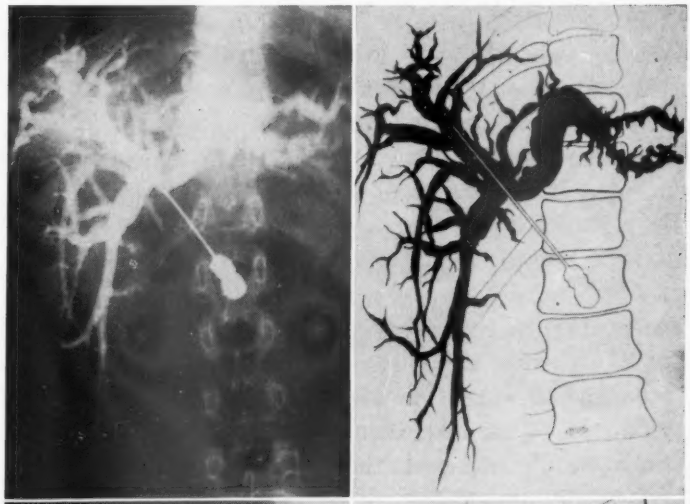


Fig. 6.

Fig. 7.

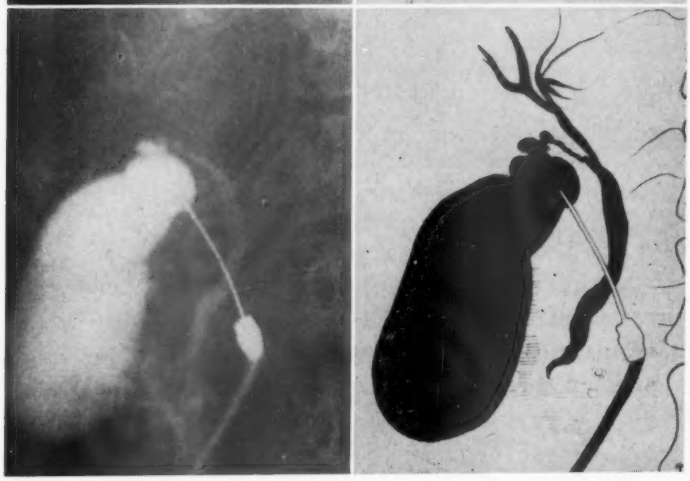


Fig. 8.

Fig. 6.—Complete obstructive jaundice caused by a stricture of the hepatic duct at the level of its bifurcation.

Fig. 7.—Partial obstruction of the distal common duct due to inflammation of the sphincter of Oddi.

Fig. 8.—Normal extrahepatic biliary tree obtained by means of transparietal vesicular cholangiography. (Jaundice caused by cirrhosis.)

Pronounced alteration in hepatic flocculation tests, as well as reduction in the prothrombin level, was observed in 40 of these patients. The prothrombin level could not be elevated above 50 per cent in 6 cases; and percutaneous transhepatic cholangiography had to be performed in one patient whose prothrombin level was as low as 19 per cent. In only 5 patients, the serum bilirubin was below 3 mg. per cent. In only one of these patients was the examination unsuccessful. The remainder of the patients had a markedly elevated serum bilirubin, the highest being 37 mg. per cent. Hepatomegaly was found in 40 patients.

Positive results were defined as the aspiration of bile through the puncture and the injection of dye into the bile ducts or the gall bladder, thus permitting radiographic visualization. In 38 cases, results were positive; and in the 8 failures, bile could not be aspirated and the dye was not injected. Of the 38 positive cases, the needle entered the bile ducts (transhepatic canalicular cholangiography) in 32 patients, and entered the gall bladder (transhepatic vesicular cholangiography) in 5. In 1 patient, the puncture was made into a cavity which contained clear watery fluid; roentgenographic examination indicated that this was an hepatic hydatid cyst (Fig. 3).

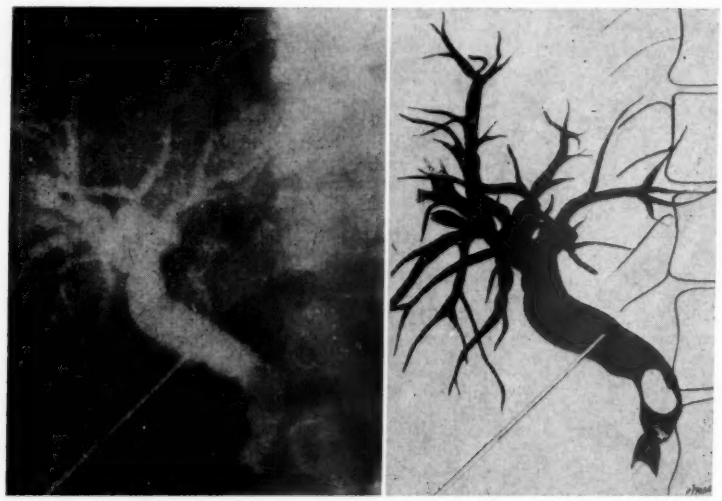


Fig. 9.—Complete obstructive jaundice caused by an impacted stone in the distal portion of the common duct. The puncture was not made through the liver's parenchyma and bile peritonitis was the result.

In the 32 cases in which transhepatic canalicular cholangiography was performed, the cause of the obstruction as determined by roentgenograms included: choledocholithiasis, 14 cases; cancer of biliary tract, 11 cases; benign stenosis of the common bile duct, 6 cases; and inflammation of the sphincter of Oddi, 1 case (Figs. 4, 5, 6, and 7).

In the 5 cases of transhepatic vesicular cholangiography, x-ray studies revealed: normal biliary tract, 2 cases; cholelithiasis, 1 case; and visualization of normal gall bladder, 2 cases.

In the 8 patients in whom puncture was unsuccessful, the clinical diagnosis obtained during surgical treatment or from later necropsies included: cholecystolithiasis and cirrhosis, 1 case; cirrhosis, 4 cases; benign stenosis of the

common duct, 1 case; choledocholithiasis and fistula between the gall bladder and common duct, 1 case; chronic pancreatitis, 1 case. Therefore, 5 of the cases were due to cirrhosis and 3 were due to extrahepatic obstruction.

In the total number of cases, a second puncture was performed in 6 patients because of either inability to reach a bile duct or poor roentgenography. In 5 patients, the second puncture was successful and both punctures failed in 1 patient.

Bacteriologic studies were carried out on the bile in 14 cases. In 8 cases of obstruction due to cancer, the cultures were negative and in the remaining 6 Klebsiella and *Escherichia coli* were found.

COMPLICATIONS

No fatalities resulted from the 52 examinations in 46 patients. Bile peritonitis developed in 1 patient, resulting from direct puncture of the common duct during cholangiography. This complication was suspected from roentgenologic evidence; the diagnosis was made early and surgical intervention was successful (Fig. 9). Shock occurred in 2 patients and both responded to treatment. The origin of this shock could not be determined, but the radiopaque solution was suspected.

Pain occurred in the right quadrant in all patients; most of it was mild, rarely severe. Occasionally the pain radiated to the right shoulder and was accompanied by spasm of the rectus muscle; at subsequent operation hours to days later neither blood nor bile could be detected in this region.

DISCUSSION

Visualization of the biliary ducts by intravenous cholangiography is possible only when the serum bilirubin is less than 3 mg. per 100 c.c. or when Bromsulphalein retention is less than 40 per cent. Percutaneous transhepatic cholangiography, therefore, is of great value in the diagnosis of patients with jaundice when the bilirubin level is higher than 3 mg. per 100 c.c. Roentgenograms taken after the injection of dye show the morphology of the biliary tract, the site of organic obstruction, and also usually the nature of the obstruction.

Percutaneous transhepatic cholangiography is also valuable in the differential diagnosis between hepatocellular and posthepatic jaundice when clinical and laboratory studies have failed.

In addition to the value of roentgenographic examination of the biliary tract, the puncture serves a further purpose in diagnosis—increased resistance when the trocar is introduced suggests cirrhosis, and decreased resistance and aspiration of blood suggests diffuse inflammation of the gland. This information assumes special importance in cases in which the puncture is unsuccessful. In the series described, percutaneous transparietal cholangiography made the differential diagnosis of jaundice in 11 patients.

In 4 patients with obstructive jaundice this method was unsuccessful in determining the etiology of posthepatic jaundice. In one case the examination was technically insufficient, but in the other 3 cases either biliary cirrhosis impeded the dilatation of the intrahepatic ducts or at the moment of the examination no biliary hypertension was present.

A positive examination depends on dilatation of the intrahepatic biliary ducts, and this in turn is related to the length of time the jaundice has been developing and on the cause of obstruction. In neoplastic obstruction, the x-ray image shows marked dilatation of the intrahepatic ducts (Fig. 5). In obstruction of benign origin the ducts are less dilated and have regular boundaries (Fig. 4). In the cases discussed, percutaneous transhepatic cholangiography is shown to be of value to the surgeon by giving him a graphic picture and permitting him to consider the surgical therapy he will employ.

SUMMARY

The technique of percutaneous transhepatic cholangiography and the results obtained in 46 cases are discussed. This examination is useful in patients with prolonged jaundice to determine the cause and the level of the obstruction, and is also of value when standard measures fail to differentiate hepatic and posthepatic jaundice.

Dye was introduced into the biliary tract of 38 of these cases (82.7 per cent). In the remaining 8 (17.3 per cent) no bile duct was punctured. In these 8 unsuccessful punctures, nonsurgical jaundice was present in 5, and therefore, in only 3 cases with posthepatic jaundice was this method unsuccessful.

In 11 cases, no exact clinical diagnosis had been made. By use of percutaneous transhepatic cholangiography, the type of jaundice was determined.

No fatalities resulted from these examinations. Complications consisted of bile peritonitis (1 case) and shock (2 cases). Pain was reported by all patients at the puncture site, occasionally accompanied by spasm of the right rectus muscle of variable intensity and duration.

Until more experience is obtained, every patient submitted to percutaneous transhepatic cholangiography should be observed in a recovery room for at least 24 hours. The surgeons should keep in mind that this type of patient is a candidate to develop intra-abdominal hemorrhage or bile peritonitis.

REFERENCES

1. Alvarez, R.: Colangiografia transabdominal, Prensa méd. argent. **39**: 935, 1952.
2. Alvarez, R.: Cholangiografia transabdominal, Prensa méd. argent. **40**: 1991, 1953.
3. Carter, F. R., and Saypol, G. M.: Transabdominal Cholangiography, J. A. M. A. **148**: 253, 1952.
4. Crismer, R.: Cholangiographie transcutaneohepatique, Acta gastroenterol. belg. **16**: 762-770, 1953.
5. De la Fuente, R., and Patillo, C.: Exploracion de las vias biliares mediante colangiografia transabdominal, Arch. Soc. cir. Chile **5**: 659, 1953.
6. De la Fuente, R., Patillo, C., Koch, W., and Mallet-Guy, P.: Exploration des voies biliares au moyen de la cholangiographie transabdominale, Lyon chir. **49**: 959, 1954.
7. Felci, L., and Felci, U.: Colangiografia transepatica percutanea, Radiol. med. **44**: 591, 1957.
8. Felci, U.: Colangiografia transepatica percutanea, Minerva med. **49**: 130, 1958.
9. Garcia Siguero, P.: Ventajas de la colangiografia transparietohepatica, Soc. argent. cirujanos bol. trab. **18**: 439-444, 1957.
10. Glenn, F., and Johnson, G., Jr.: Intravenous Cholangiography: Correlation With Operative Findings in 72 Patients, Surg. Gynec. & Obst. **105**: 473, 1957.
11. Goñi Moreno, I.: Colangiografía por puncion percutánea, Bol. Acad. argent. cir. **37**: 335, 1953.

12. Halligan, E. L., and Farid, R.: Transcutaneous Hepatic Cholangiographic Study: Its Importance in the Diagnosis and Management of Biliary Disease, J. Internat. Coll. Surgeons **31**: 145, 1959.
13. Kidd, H. A.: Percutaneous Transhepatic Cholangiography, A. M. A. Arch. Surg. **72**: 262, 1956.
14. Lasala, A.: La colangiografía clínica puede reemplazar a la qurúrgica, Prensa méd. argent. **44** (33): 2598, 1957.
15. Leger, L.: Cholangiographie et drainage biliare par ponction transhepatique, Presse méd. **60**: 936, 1952.
16. Leger, L., Zara, M., and Wargnier, M.: Cholangiographie et drainage biliare par ponction transparietohepatique, Arch. mal. app. digestif. **42**: 967, 1953.
17. Magistocchi, L. A.: Colangiografía transcutánea, Bol. Soc. cir. Buenos Aires **37**: 666, 1953.
18. Mandl, F.: Die transcutanen Cholangiographie bei ikterischen Patienten, Chirurg. **27**: 241-243, 1956.
19. Mandl, F.: Weitere Entwicklung der transcutanen und transhepaten Cholangiographie bei Relaparotimiun wegen erfolgloser Operationen an ikterischen Kranken, Chirurg. **28**: 549-550, 1957.
20. Meerof, M.: ¿Es la colangiografía T. P. H. una técnica "inadmissible"? Prensa méd. argent. **44**: 3787-3791, 1957.
21. Michelini, R.: Colangiografía transabdominal, Prensa méd. argent. **40**: 1094, 1953.
22. Nurick, A. W.: Percutaneous Transhepatic Cholangiography in the Diagnosis of Obstructive Jaundice, Brit. J. Surg. **41**: 27, 1953.
23. Remolar, J., Katz, S., Ribak, B., and Pellizari, O.: Percutaneous Transhepatic Cholangiography, Gastroenterology **31**: 39-46, 1956.
24. Richieri, A.: La colangiografía T. P. H. y sus accidentes, Prensa méd. argent. **44**: 2865, 1957.
25. Richieri, A.: La colangiografía T. P. H. y sus accidentes, Prensa méd. argent. **44**: 2799, 1957.
26. Royer, M.: Nonsurgical Cholangiography; Technic, Semana méd. **2**: 926, 1947.
27. Royer, M.: Transhepatic Laparoscopic Cholangiography, Prensa méd. argent. **41**: 1058-1062, 1954.
28. Royer, M.: La colangiografía laparoscópica, El Ateneo, B. A., 1952.
29. Royer, M.: La colangiografía T. P. H. bajo control radioscópico, Día méd. **28**: 1889, 1956.
30. Royer, M.: La cholangiographie transparieto-hepatique sous controle radioscopique, Presse méd. **64**: 976-977, 1956.
31. Seldinger, S. I.: A Simple Method of Catheterization of the Spleen and Liver, Acta radiol. **48**: 93-96, 1957.
32. Sobredo, E., and Garcia Siguero, P.: Colangiografía transparietohepatica, Orientación méd. B. A. **3**: 119, 1954.
33. Varela Fuentes, B., Bertoni, C., and Polero, J.: La colangiografía por punción hepática, Prensa méd. argent. **44**: 2873-2883, 1957.
34. Wilks, A. E., Valenzuela, D., and Schaposnik, F.: La colangiografía transparieto-hepática, Prensa méd. argent. **45**: 2277-2279, 1958.
35. Wise, R. E.: Roentgenographic Diagnosis of Biliary Tract Disease, Postgrad. Med. **21**: 531-542, 1957.
36. Zollinger, Robert M., and Williams, R. D.: Surgical Aspects of Jaundice, SURGERY **39**: 1016, 1956.

STENOSIS OF THE SPHINCTER OF ODDI

A Clinicopathologic Review of 50 Cases

Theodor B. Grage, M.D., Paul H. Lober, M.D., Kamil Imamoglu, M.D., and Owen H. Wangensteen, M.D., Minneapolis, Minn.

(From the Departments of Surgery and Pathology, University of Minnesota Medical School)

TRANSDUODENAL exploration of the common bile duct and division of the sphincter of Oddi have become acceptable surgical procedures in the management of certain benign obstructive lesions of the choledochoduodenal junction. As yet, there is no general agreement concerning the exact nature and significance of these conditions and their etiology is poorly understood.

During the past few years, biopsies from the terminal common bile duct in 50 patients requiring sphincterotomy have been examined. In addition, the region of the sphincter of Oddi from 50 unselected autopsies has been studied histologically. It is the purpose of this report to describe some of the findings at operation associated with stenosis of the distal common bile duct, and to indicate that only in a certain number of patients is it possible to find and demonstrate evidence of pathologic changes by biopsy. An attempt will be made to correlate the microscopic alterations with the clinical condition of the patient and the findings at operation.

HISTORICAL BACKGROUND

In tracing some of the historic highlights pertaining to the surgical aspects of the pathology of the choledochoduodenal junction, it is interesting to find that in 1884 Carl Langenbuch,[13] the "father of biliary surgery," only 2 years after reporting the first successful removal of a gall bladder, suggested transduodenal division of the "diverticulum" of Vater in cases of cicatricial stenosis from chronic inflammation.[14] McBurney,[18] in 1892, extracted an impacted stone from the ampulla after sphincterotomy and reported 6 patients thus treated in 1896. Kocher,[12] in 1894, used the same approach for removal of stones from the distal common bile duct, but he felt that the cut edges of the biliary and duodenal mucosa should be approximated and called this sphincteroplasty a "choledochoduodenostomia interna." Summers,[22] in 1900, treated an 18-year-old jaundiced girl, after exploration of the common bile duct had revealed no stone, by a choledochoduodenostomy for an "evident stricture near the duodenal end of the duct."

The "common channel" theory received its impetus from Opie's report[20] in 1901, which implicated an impacted stone in the ampulla as the cause of acute pancreatitis. Archibald, in 1913, suggested sphincterotomy in the treatment of pancreatitis as the best means to lower biliary pressure.[1] Doubilet and

Received for publication Dec. 13, 1959.

Mulholland,[7, 8] after extensive experience with this procedure, believe it to be the treatment of choice for recurrent pancreatitis.

Berg[2] treated 2 patients with "contracture" of the sphincter of Oddi by dilatation. He believed that dysfunction of the sphincter muscle was primarily responsible for the genesis of biliary stones and would lead to secondary organic changes within the sphincter. Westphal,[24] in 1923, on the basis of numerous clinical studies, expressed a similar view and speaks of a hypertonic and hypotonic motility neurosis of the biliary tract.

In 1928 del Valle[6] treated several patients for a "fibrotic odditis" and believed one should be able to make this diagnosis preoperatively. By 1933, Strauss[21] was able to report 29 cases of "chronic biliary stasis" caused by inflammation and hypertrophy at the ampulla of Vater. However, not until very recently has the existence of benign obstructive lesions at the choledochoduodenal junction and its successful management by sphincterotomy received more general acceptance.[3, 4, 15, 16]

Mallet-Guy[17] stresses the use of radiomanometry in the diagnosis of disorders of the sphincter of Oddi. Other authors rely more upon the use of various-sized probes in assessing the degree of patency of the terminal common bile duct.[3, 16]

Mahorner[16] reported 40 cases of stenosis of the sphincter of Oddi in 445 operations upon the biliary tract and Fahrlander[9] reported 87 cases of inflammatory disease of the papilla of Vater in 533 patients operated upon for biliary disease.

CLINICAL DATA

Biopsies from the periampullary region were obtained from 50 patients in whom a sphincterotomy had been performed at the University of Minnesota Hospitals in the period from January, 1955, to April, 1959, with the exception of 1 patient operated upon in 1949. Of these, 31 were females and 19 males. Their ages ranged from 14 to 77 years, with 40 patients over the age of 40 and 22 patients over the age of 60.

The most frequent complaint was epigastric or right upper abdominal pain, usually colicky and intermittent, and at times radiating to the back and interscapular region. Evidence or a history of jaundice was obtained from 14 patients.

Radiologic visualization of the extrahepatic biliary tract was obtained by oral cholecystography and particularly by intravenous cholangiography in those patients suspected of having disease in the biliary duct system.

At operation, assessment of the patency and size of the choledochoduodenal junction by means of various-sized probes was an integral part of the common bile duct exploration in almost every patient. Inability to pass a 3 mm. Bakes' dilator through the terminal common bile duct without undue force was considered evidence of stenosis and indication for transduodenal sphincterotomy. In 5 patients, sphincterotomy was performed even though a 3 mm. probe had been passed with some difficulty.

Direct measurement of pressure within the common bile duct during operation in a few patients did not provide sufficient additional information to

warrant its routine use. A normal outflow pressure may occur in the presence of a stenotic papilla. Similar experiences have been recorded by others.[5, 11] Passing a probe appears to be more reliable as a test of a patulous and nonstenotic papilla. In fact, in this particular, it probably gives more information than does the cholangiogram.

Irrigation of the distal common bile duct by means of a small rubber catheter with a local anesthetic (5 per cent Cyclaine) to overcome spasm of the sphincter of Oddi also has been employed. Of 7 patients in this group thus treated, no effect was noted in 5, and in 2 patients only a 4 mm. probe could be passed with some difficulty following instillation of the local anesthetic.

Operative cholangiography, either through a choledochotomy or the cystic duct stump, was resorted to in 11 instances.

The technique of sphincterotomy will not be described in detail. Briefly, it was performed through a short transverse duodenotomy and, with the papilla elevated by the dilator, the sphincter was incised sufficiently to permit passage of a 6 to 8 mm. probe. A piece of tissue corresponding to the length of the sphincterotomy was removed for study.

In 14 patients concomitant surgical procedures were performed and are summarized in Table I. The association of peptic ulcer and stenosis of the sphincter of Oddi is thought to be incidental in this group and probably reflects the type of patients submitted to surgery rather than a causal relationship.

TABLE I. CONCOMITANT OPERATIVE PROCEDURES IN 15 PATIENTS TREATED FOR STENOSIS OF THE SPHINCTER OF ODDI

DIAGNOSIS AND PROCEDURE	NO. OF CASES
Subtotal gastric resection for peptic ulcer	11
Subtotal colectomy for diverticulitis	1
Heller myotomy for esophageal achalasia	1
Repair of hiatal hernia	1
Repair of ventral hernia	1
Total	15

From a clinical point of view, the patients fall into 3 groups: (1) patients with no previous biliary surgery, (2) patients with recurrent symptoms after previous operation upon the biliary tract, and (3) patients operated upon for recurrent pancreatitis.

Group 1.—Of 29 patients in this category, 13 were found to have a dilated common bile duct, which contained stones in 9 patients. In 4 instances stones were lodged within the ampulla. A diagnosis of pancreatitis was made in 1 case. In view of the demonstration that in a certain number of patients with cholelithiasis a stenosis of the sphincter of Oddi exists,[10] inspection of the papilla has become a routine operative procedure on one of the surgical services for all patients with cholelithiasis in whom a 3 mm. dilator cannot be passed with ease. Biopsies from 9 such patients, in whom the conventional indications for choledochotomy did not prevail and in whom a sphincterotomy was deemed necessary, were available for study.

Group 2.—Of 17 patients with recurrent symptoms of biliary tract disease, 13 had undergone a cholecystectomy from 6 months to 25 years previously, and

in 4 patients a cholecystostomy had been performed more than 20 years before. The common bile duct had been explored for stones in 5 of them. Three patients had been operated upon more than once for their symptoms. At the time of admission, 5 patients were jaundiced and 2 more gave a history of jaundice. The findings at operation in these 17 patients are summarized in Table II. The only positive findings in 3 of these patients were a dilated common bile duct and a stenosis at the choledochoduodenal junction. In 3 of the 5 patients with choledocholithiasis, a stone was impacted in the ampulla.

TABLE II. OPERATIVE FINDINGS IN 17 PATIENTS WITH PREVIOUS SURGERY OF THE BILIARY TRACT

	NO. OF CASES
Chronic cholecystitis and cholelithiasis	4
Long cystic duct remnant	2
Stones in common bile duct	5
Evidence of chronic pancreatitis	6
Recurrent duodenal ulcer	1
Subhepatic abscess	1
Cholecystoduodenal fistula	1
Dilatation of common bile duct	15

Group 3.—Of 4 patients operated upon for recurrent pancreatitis, 2 were found to have a stricture at the pancreaticocholedochal junction. One patient had undergone a cholecystectomy 2 years previously. In another patient, 2 months prior to the sphincterotomy, a cholecystostomy had been performed with the first attack of acute pancreatitis.

GROSS AND MICROSCOPIC OBSERVATIONS OF THE CHOLEDOCHODUODENAL JUNCTION

For the purpose of this study, 50 biopsy specimens taken from the duodenal papilla in patients requiring sphincterotomy and 50 specimens taken from autopsy cases selected at random were examined.

Biopsy Material.—

Gross observations: Descriptions of the gross appearance and abnormal features of the terminal common bile duct were obtained from the operative records of these patients. In many instances, the description consisted only of a short statement that a 3 mm. dilator could not be passed or that the sphincter appeared stenotic. However, in others, more detailed information was available.

In 5 patients, the papilla was unusually prominent and thick with marked narrowing of the lumen. All 5 patients were found to have evidence of chronic pancreatitis. In 2 of these, preoperative upper gastrointestinal studies revealed a persistent filling defect in the second part of the duodenum (Fig. 1).

In 3 patients the stenosis was confined to the extreme tip of the papilla. A 3 mm. dilator could be passed through most of the intramural portion of the terminal common duct before encountering a pin-point stricture at the very end of the papilla.

In 14 patients, the stricture extended from the tip of the papilla for a varying distance toward the junction of the common bile duct with the muscular wall of the duodenum. Usually the stenosis was most marked at the tip of the

papilla, as evidenced by the ability to pass a 3 or 4 mm. dilator after a short division of the papilla. To accommodate a 6 or 7 mm. probe, a further extension of the sphincterotomy was necessary in most of these 14 patients.

In 3 patients, the stenosis was confined mainly to the deeper portions of the intramural duct, near its junction with the duodenal musculature. In no instance was it necessary to extend the sphincterotomy beyond the wall of the duodenum.

Microscopic findings: The biopsy specimens included in all cases the anterior lip of the orifice of the ampulla with a small portion of the overlying duodenal mucosa. Variable amounts of the wall of the ampulla and the intramural bile duct were included in the biopsy. During the time this series was collected. A biopsy of the region of the sphincter of Oddi was performed in 27 additional cases, but the amount of tissue available was considered inadequate for inclusion in this study.

Fig. 1.—Unusually large and prominent papilla of Vater seen on gastrointestinal series. (Thal, Alan P., Goott, Bernard, and Margulis, Alexander R.: Sites of Pancreatic Duct Obstruction in Chronic Pancreatitis, Ann. Surg. 150: 49-56, 1959. Reproduced with permission of authors and publishers.)

Several microscopic sections of each biopsy were prepared and stained with hematoxylin and eosin and with Masson's trichrome stain. The latter stain is particularly suitable for demonstrating changes in fibrous tissue. Although serial sectioning was not attempted, generally all of the biopsy tissue was sectioned. The biopsies were examined without previous knowledge of the clinical history, and a notation was made of any abnormal features.

The histologic examination was made particularly for evidences of inflammation or fibrosis in the terminal bile duct or ampulla. The subepithelial layer of the bile duct, especially, was examined for inflammatory cellular infiltrations and increased fibrous tissue. The latter might be manifested by increased thickness of the lamina propria, separation of the muscle bundles, and by separation, compression, or disappearance of mucous glands. Cellularity and fiber thickness of the collagenous tissue might also be altered. Dilatation or hyperplasia of the glands in the papilla was sought. Possible hypertrophy of the muscle layers was found to be impossible to assess in the material examined.

On the basis of the microscopic examination, 18 of the biopsies were found to be without recognizable alterations.

Scattered lymphocytes or plasma cells, occasionally as small aggregates, within the papilla or terminal duct wall were seen in 18 of the biopsies. Two showed an infiltrate of polymorphonuclear leukocytes in the papilla. Along with the cellular infiltration, there were dilated capillaries adjacent to the duct lumen in 7 cases.

Some dilatation of the paraductal glands, particularly at the tip of the papilla, was noted in 7 biopsies (Fig. 2). A small amount of squamous metaplasia was found in 1 case (Fig. 3), and 1 ampulla contained a small pyogenic granuloma (Fig. 4).

Nineteen of the biopsies were believed to show some alteration in the amount of fibrous tissue at the choledochoduodenal junction. In 17 of these cases, there were changes which indicated slight increases in fibrous tissue. These consisted of localized patches in which the mucous glands of the intramural duct appeared surrounded by layers of collagenous fibers, and the acini were separated or reduced in size (Fig. 5). In a few, the muscle fibers in local areas seemed separated by coarser strands of collagen. These changes were minimal and in no case warranted a histologic diagnosis of fibrosis of the sphincter of Oddi. However, in 2 of these 19 biopsies, there appeared to be a definite increase in the thickness of the subepithelial layer of the intramural bile duct. It appeared that the glandular elements were displaced and the muscle bundles separated. The fibrous tissue itself was not remarkably cellular (Fig. 6).

There was no correlation in either of these groups having alteration of the fibrous tissue, with the other changes noted. Approximately equal proportions of those with or without fibrous changes showed inflammatory cellular infiltration or glandular dilatation. The specimen containing the pyogenic granuloma was thought to show a slight increase in fibrous tissue, while that exhibiting the squamous metaplasia did not.

Autopsy Material.—

It was hoped that the larger specimens obtained from 50 unselected autopsies would be helpful in establishing the normal variations in structure and in orienting the biopsy specimens (Figs. 8 and 9).

Gross findings: In 31 cases the biliary tract was found to be grossly normal. The gall bladder contained stones in 13 instances and in 6 cases the gall bladder had been removed at some time in the past. The common bile duct was dilated in 3 cases and in 1 instance a diagnosis of chronic pancreatitis was made.

Grossly, no particular abnormalities of the choledochoduodenal junction were noted in any of the 50 autopsies. In 25 cases, the actual size of the terminal common bile duct was measured by means of various-sized dilators. A 7 mm. probe could be passed in only 2 patients, both of whom had no biliary disease. In 11 cases, a 5 mm. probe was passed without difficulty. In 4 of these, the gall bladder either contained stones or had been removed previously because of stones. In 12 instances, the terminal common duct admitted only a 3 mm.

Fig. 2.

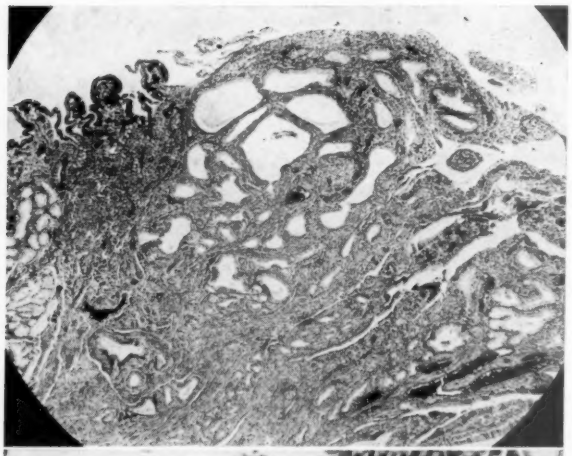


Fig. 3.

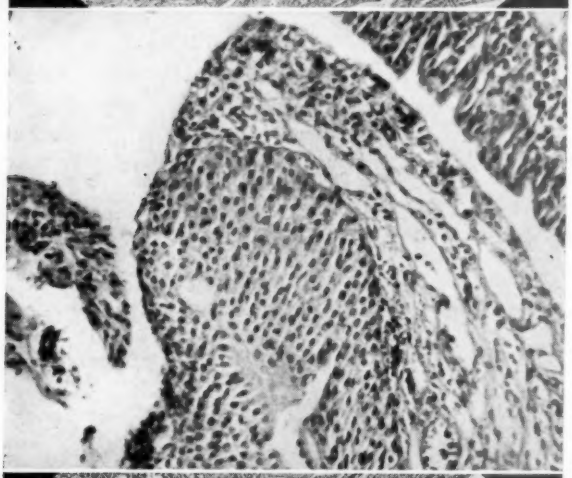


Fig. 4.

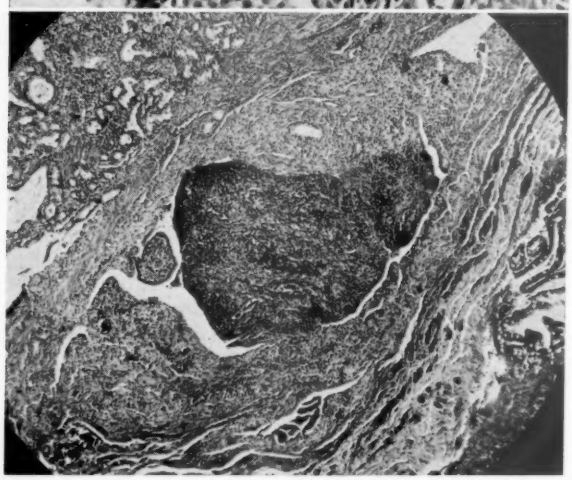


Fig. 2.—Photomicrograph of biopsy of papilla showing dilatation of paraductal glands. (Masson's trichrome; ×60; reduced ⅓.)

Fig. 3.—Photomicrograph showing small area of squamous metaplasia from tip of papilla. (Hematoxylin and eosin; ×300; reduced ⅓.)

Fig. 4.—Photomicrograph of biopsy showing polypoid mass of fibrous tissue and capillaries with inflammatory cells resembling a pyogenic granuloma. (Masson's trichrome; ×35; reduced ⅓.)

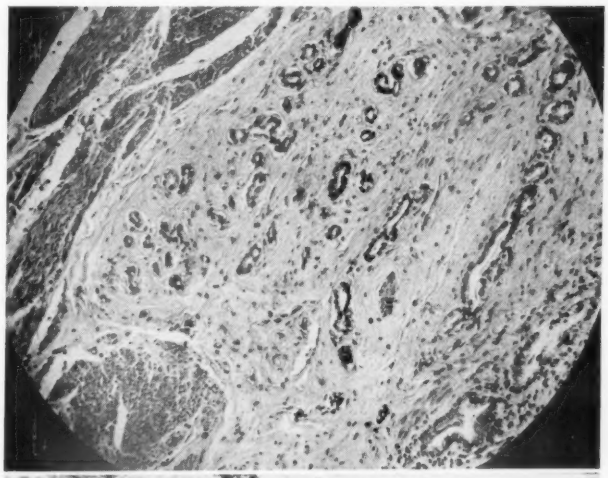


Fig. 5.

Fig. 6.

Fig. 7.

Fig. 5.—Photomicrograph of periampullary region showing minimal fibrous changes. The glandular acini are reduced in size and surrounded by proliferating fibrous tissue. (Masson's trichrome; ×135; reduced ⅓.)

Fig. 6.—Photomicrograph of biopsy of papilla which shows diffuse fibrous changes. (Masson's trichrome; ×60; reduced ⅓.)

Fig. 7.—Photomicrograph of autopsy section from terminal common bile duct at junction with pancreatic duct. Note normal distribution of paraductal glands. (Masson's trichrome; ×35; reduced ⅓.)

dilator. The gall bladder contained stones or had been removed previously in 8 of these 12. As can be seen from Table III, the larger dilators could be passed relatively more often in cases without biliary disease than in those with biliary disease.

TABLE III. AUTOPSY OBSERVATIONS ON SIZE OF PAPILLA OF VATER

SIZE OF BAKES' DILATOR THAT READILY PASSED THROUGH PAPILLA	NO. OF PATIENTS WITH BILIARY DISEASE	NO. OF PATIENTS WITHOUT BILIARY DISEASE
3 mm.	8	4
5 mm.	4	7
7 mm.	—	2

Fig. 8.

Fig. 9.

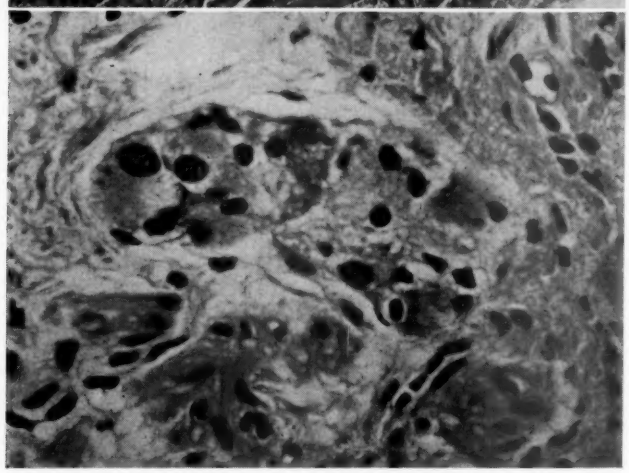


Fig. 8.—Photomicrograph of autopsy section of periampullary region showing normal association of glands and muscle bundles. (Masson's trichrome; ×70; reduced ⅓.)

Fig. 9.—Photomicrograph which shows small group of ganglion cells within muscle of terminal common bile duct wall from a patient with diffuse fibrosis of periampullary region. (Hematoxylin and eosin; ×660; reduced ⅓.)

Microscopic observations: The autopsy sections were taken longitudinally and included the anterior wall of the entire intramural portion of the duct, and

often a segment of the terminal bile duct proximal to the duodenal wall. The autopsy sections were stained and reviewed in the same manner as the biopsies.

Examination of the 50 autopsy specimens showed considerable variation in the structure of the intramural bile duct. The tip of the papilla showed an increased number of glands, which were somewhat dilated, in 2 cases. Within the lining of the duct, there were a few foci of lymphocytes in 5 cases. In 2 of these, the gall bladder contained stones. No other histologic changes were found.

Changes corresponding to those seen in the 17 biopsies with minimal fibrous changes were found in 3 specimens from the autopsies. Only one of these specimens came from a patient with gallstones.

No cases were found that corresponded with the more definite fibrous alterations of the biopsies. The microscopic changes noted in the autopsy group are summarized in Table IV.

TABLE IV. MICROSCOPIC CHANGES AT THE TERMINAL COMMON BILE DUCT IN 50 UNSELECTED AUTOPIES

		MICROSCOPIC CHANGES			
	NO CHANGE	MINIMAL FIBROUS CHANGES	DIFFUSE FIBROSIS	INFLAMMATORY INFILTRATE	DILATED GLANDS
Total no. (50)	41	3	—	5	2
a. with biliary disease (19)	16	1	—	2	—
b. without biliary disease (31)	25	2	—	3	2

The larger amount of tissue for examination in the autopsy specimens made a better evaluation of duct wall changes possible. It was evident from the autopsy material that sections taken more proximally in the duct would show greater thickness of the lamina propria and relatively fewer glands, although the muscle was generally also more prominent in this region. The junction of the pancreatic duct was close to the papilla occasionally, and appeared to contribute some thickness to the wall.

OBSERVATIONS PERTAINING TO THE NEURAL ELEMENTS OF THE DISTAL COMMON BILE DUCT

The question has been raised whether abnormalities of the sphincter of Oddi might not be due to absence of neural elements in the sphincter muscle.[23] However, examination of the autopsy and biopsy specimens in almost every instance revealed ganglion cells scattered singly or in small groups of 2 to 3 cells within the muscle of the terminal common duct, regardless of the microscopic pathology (see Fig. 9). In addition, in 14 of 30 autopsy specimens examined for this purpose, collections of from 6 to 30 ganglion cells were found within the tissue surrounding the common bile duct at its junction with the duodenum. Although the exact role of the sympathetic and parasympathetic nervous system in the function of the sphincter of Oddi has not been elucidated, the presence of these ganglion cells suggests some measure of control of this mechanism by the autonomic nervous system.

COMPARISON OF THE MICROSCOPIC FINDINGS WITH THE GROSS PATHOLOGY

Review of the clinical data in the biopsy series showed no definite correlation between the histologic changes noted and the various manifestations of biliary tract disease (Table V). Minimal patchy fibrosis occurred with approximately equal frequency in patients in whom the common duct was explored for "conventional" indications, and in those in whom a narrowed terminal common bile duct was found subsequent to a "routine" examination of the papilla. The histologic changes in the periampullary tissue in patients operated upon for the first time did not differ materially from those observed in patients operated upon for recurrent symptoms. The microscopic findings in the biopsies from patients with evidence of pancreatitis or peptic ulcer were similar to those found in the patients who did not have these disorders.

TABLE V. MICROSCOPIC CHANGES IN BIOPSIES OF THE SPHINCTER OF ODDI IN VARIOUS CLINICAL GROUPS

	MICROSCOPIC CHANGES					
CLINICAL GROUP	NO CHANGE	MINIMAL FIBROUS CHANGES	DIFFUSE FIBROSIS	INFLAMMATORY INFILTRATE	DILATED GLANDS	OTHER
I. Total no. of cases (50)	18	17	2	18	7	2
II. Primary biliary operation (29)	10	12	—	10	5	2
a. Jaundice common bile duct stones common bile duct dilatation (20)	8	8	—	6	3	
b. "Routine" inspection of papilla (9)	2	4	—	4	2	2
III. Secondary biliary operation (17)	6	4	2	7	1	
a. Evidence of pancreatitis (6)	1	1	1	4	—	
b. No pancreatitis (11)	5	3	1	3	1	
IV. Recurrent pancreatitis (4)	2	1	—	1	1	

Comparison of the histologic changes with the gross findings at the terminal common bile duct reveals a relatively higher incidence of fibrous tissue alteration in the biopsies from patients with the most stenotic papilla, as manifested by the inability to pass even a 2 mm. dilator (Table VI). Of the 7 specimens from patients in whom stones were lodged within the ampulla, 2 showed minimal fibrous changes and an inflammatory infiltrate was noted in 5. The biopsies from the 5 patients with prominent and large papillas revealed minimal fibrous changes in 1, an inflammatory infiltrate in 2, and no apparent histologic changes in the remaining 2 biopsies.

Minimal fibrous changes were noted in 2 of the 3 biopsies from the patients in whom the stricture was confined to the tip of the papilla.

TABLE VI. COMPARISON OF THE MICROSCOPIC CHANGES WITH THE GROSS FINDINGS AT THE TERMINAL COMMON BILE DUCT

GROSS FINDINGS	MICROSCOPIC CHANGES					
	NO CHANGE	MINIMAL FIBROUS CHANGES	DIFFUSE FIBROSIS	INFLAMMATORY INFILTRATE	DILATED GLANDS	OTHER
I. Total no. (50)	18	17	2	18	7	2
II. Size of Bakes' dilator that did not readily pass through papilla						
a. 2 mm. (6)	—	4	—	3	—	—
b. 3 mm. (25)	10	9	—	6	5	2
c. 4 mm. (5)	4	—	1	—	—	—
d. 7 mm. (1)	—	1	—	1	—	—
e. not stated (6)	3	1	1	1	1	—
III. Stone impacted at ampulla (7)	1	2	—	5	1	—
IV. Large papilla (5)	2	1	—	2	—	—

Of the 14 patients with a stricture involving most of the length of the papilla, minimal fibrous changes were found in 5 biopsies, a diffuse fibrosis in 1, and a lymphocytic infiltrate in 2 specimens. Even though the terminus of the common bile duct in 5 of these 14 patients was described as extremely stenotic and fibrotic, 4 of these 5 biopsies were without apparent alterations histologically.

Minimal fibrous changes were noted in only 1 of the 3 biopsies from patients in whom the stricture was located in the deeper portion of the papilla.

DISCUSSION

In this group of 50 cases of clinical stricture of the terminal common bile duct, any alteration in fibrous tissue could be demonstrated in less than one half of the biopsies studied. If one includes the cases with an inflammatory infiltrate, dilatation of the paraductal glandular tissue and such lesions as pyogenic granuloma and squamous metaplasia, the incidence of histologic changes rises to approximately 64 per cent.

It appears that biliary tract disease with stenosis of the choledochoduodenal junction may be present in many cases without any evident histologic change in the periampullary tissue. In some other cases there are minor histologic changes, which, however, may also be found in some autopsy specimens from patients without evidence of biliary tract disease.

There remain 2 cases of stenosis in which there are definite fibrotic changes in the periampullary tissue. The significance of these in relation to the stenosis is not clear from this study, since equally severe biliary tract disease may be present without these changes in the majority of cases.

In view of these findings, it would appear that a nonspecific clinical term, such as stenosis, would be more correct in designating benign obstructive lesions at the choledochoduodenal junction than such terms as fibrosis of the sphincter of Oddi,[3] sclerosing Odditis,[6] or papillitis.

Division of the sphincter of Oddi is not without hazard. Two patients developed acute hemorrhagic pancreatitis postoperatively, with a fatal outcome

in 1. In 1 patient, a stricture at the terminal bile duct recurred, which was not relieved after repeat sphincterotomy, and a choledochoduodenostomy was ultimately necessary. Cholangitis due to *Staphylococcus aureus* occurred in 1 patient.

Although the immediate result of operation has been good in nearly all patients as judged by relief of their symptoms, prolonged observation will be necessary to evaluate properly the result of sphincterotomy.

SUMMARY

A series of 50 biopsies of the duodenal papilla from patients in whom sphincterotomy was performed have been studied histologically, and the clinical histories and operative findings in these cases reviewed. Specimens of periampullary tissue from 50 unselected autopsy cases were used for comparison.

No abnormalities were seen in 18 biopsies. Eighteen showed inflammatory cellular infiltration, 17 showed minimal fibrous changes, and 2 showed more diffuse fibrous tissue increase. Glandular dilatation was seen in 7, whereas squamous metaplasia and pyogenic granuloma were seen in 1 case each.

No definite correlation between the various manifestations of biliary tract disease and the histologic changes were found. A consistent histologic basis for the stenosis at the choledochoduodenal junction in these cases is not apparent from this study.

REFERENCES

1. Archibald, Edward: Ideas Concerning the Causation of Some Cases of Pancreatitis, Canad. J. M. & S. **33**: 263-268, 1913.
2. Berg, John: Studien über die Function der Gallenwege unter Normalen und Gewissen Abnormalen Verhältnissen, Acta chir. scandinav. **55**: (Suppl. II): 1-185, 1922.
3. Cattell, Richard B., and Colcock, Bentley P.: Fibrosis of the Sphincter of Oddi, Ann. Surg. **137**: 797-806, 1953.
4. Cattell, Richard B., Colcock, Bentley, P., and Pollack, Jerome L.: Stenosis of the Sphincter of Oddi, New England J. Med. **256**: 429-435, 1957.
5. Dahl-Iversen, A., Sorenson, H., and Westengaard, E.: Pressure Measurement in the Biliary Tract in Patients After Cholecystolithotomy and in Patients With Dyskinesia, Acta chir. scandinav. **114**: 181-190, 1957.
6. Del Valle, Delfor: Patologia del esfinter de Oddi, Rev. brasil de med. e pharm. **4**: 479-489, 1928.
7. Doubilet, Henry, and Mulholland, John H.: Surgical Treatment of Recurrent Pancreatitis by Endocholedochal Sphincterotomy, Surg. Obst. & Gynec. **86**: 295-306, 1948.
8. Doubilet, Henry, and Mulholland, John H.: Eight Year Study of Pancreatitis and Sphincterotomy, J. A. M. A. **160**: 521-528, 1956.
9. Fahrländer, H.: Über die Klinik der Erkrankungen der Extrahepatischen Gallenwege, Gastroenterologia **87**: 248-267, 1957.
10. Imamoglu, Kamil, Perry, John F., and Wangensteen, Owen H.: Experimental Production of Gallstones by Incomplete Stricture of the Terminal Common Bile Duct, Surgery **42**: 623-630, 1957.
11. Kaiser, E.: Postoperativ Veränderungen des Normalen und des Pathologischen Choledochus, Helvet. chir. acta **25**: 5-25, 1958.
12. Kocher, T.: Cited by Hans Kehr *in* Chirurgie der Gallenwege. Neue Deutsche Chirurgie, Stuttgart, 1913, Verlag von Ferdinand Enke, vol. 8.
13. Langenbuch, Carl: Ein Fall von Extirpation der Gallenblase wegen chronischer Cholelithiasis, Heilung, Berl. klin. Wchnschr. **19**: 725-727, 1882.
14. Langenbuch, Carl: Einiges über Operationen am Gallensystem, Berl. klin. Wchnschr. **21**: 809-811, 826-829, 1884.
15. Mahorner, Howard: Combined Supraduodenal and Transduodenal Exploration of the Common Bile Duct, Ann. Surg. **129**: 766-774, 1949.
16. Mahorner, Howard: Supra and Transduodenal Exploration of the Common Bile Duct, Am. J. Gastroenterol. **32**: 182-189, 1959.

17. Mallet-Guy, P.: JeanJean, R., and Feroldi, J.: La maladie du sphincter d'Oddi, Lyon chir. **40**: 553-586, 1945.
18. McBurney, Charles: Removal of Biliary Calculi From the Common Duct by the Duodenal Route, Ann. Surg. **28**: 481-486, 1898.
19. Meier, A. L., and Schweingruber, B.: Über die Indikation zur Duodenotomie und Sphincterotomie in der Gallenchirurgie, Helvet. chir. acta **25**: 317-320, 1958.
20. Opie, Eugene L.: The Etiology of Acute Hemorrhagic Pancreatitis, Bull. Johns Hopkins Hosp. **12**: 182-188, 1901.
21. Strauss, Alfred A., Strauss, Siegfried F., Crawford, Robert A., and Strauss, Hermann A.: Chronic Biliary Stasis. Treatment by Choledochoduodenostomy and Gastro-enterostomy, J. A. M. A. **101**: 1365-1370, 1933.
22. Summers, John E.: A Contribution to the Surgery of the Common Bile Duct: Report of a Case of Choledochoenterostomy, J. A. M. A. **34**: 592-593, 1900.
23. Wangensteen, Owen H.: A Physiologic Operation for Mega-esophagus (Dystonia, Cardiospasm, Achalasia), Ann. Surg. **134**: 301-318, 1951.
24. Westphal, Karl: Muskelfunction, Nervensystem und Pathologie der Gallenwege. III. Die Motilitätsneurose der Gallenwege und ihre Beziehungen zu deren Pathologie, zur Stauung, Entzündung, Steinbildung, u. s. w., Ztschr. f. klin. Med. **96**: 95-150, 1923.

THE ROLE OF INCOMPLETE PANCREATIC DUCT OBSTRUCTION IN THE ETIOLOGY OF PANCREATITIS

ROBERT E. HERMANN, M.D., AND JOHN H. DAVIS, M.D., CLEVELAND, OHIO

(From the Department of Surgery, Western Reserve University and University Hospitals of Cleveland

FOR half a century much effort and thought has been devoted to the etiology of acute and chronic pancreatitis. Through this effort, it has become accepted that no single etiological factor explains all cases. Those factors which have been held responsible and most extensively studied are reviewed in excellent recent summaries.[2, 13] They are (1) reflux of bile into the pancreatic duct via a "common channel," (2) obstruction to the secretion of pancreatic juice, (3) infection, either secondary to the reflux of infected material into the pancreatic duct or from systemic infection elsewhere, (4) trauma to the gland, (5) vascular ischemia of the pancreas, (6) circulating trypsin or proteolytic enzymes, and (7) an allergic response to previous sensitization.

The great majority of cases of pancreatitis seen clinically are believed to be a result of the first 2 etiological factors, biliary reflux or obstruction of the pancreatic duct. The other factors are probably of significance in a minority of cases.

Of these 2 factors responsible for most cases, the first, that of reflux of bile up the pancreatic duct, has received the most intensive study. Since the original observations of Opie in 1901, much experimental work and many anatomic studies have been made to clarify the role of the "common channel." From this work, it is known that bile refluxed up the pancreatic duct will cause hemorrhage and necrosis of the pancreas. However, 2 objections to this theory have long prevented its acceptance as the etiological factor involved in all cases.

The first and most important objection is that a common channel does not exist anatomically in all patients. Extensive autopsy dissection studies by various groups have shown that a true ampulla, large enough for a potential common channel to be formed behind the papillary orifice, exists in only 20 to 30 per cent of all persons.[6] However, other studies based on occlusion of the papilla and instillation of fluid into the common bile duct have shown reflux to be possible in 60 to 70 per cent of patients.[4] This is the greatest number of patients in whom biliary reflux could be anatomically possible.

The second objection to the common channel theory, now largely resolved, is on the basis of comparative pressures in the biliary and pancreatic ductal systems and the high pressures required to reflux bile into the pancreas. These studies have shown that pressures in the biliary system are lower than those in the pancreatic system, both during the secreting and the resting state.[7] Thus, hydrodynamic flow would be from pancreatic to biliary system, not vice versa.

Received for publication Jan. 25, 1960.

During vomiting and straining, however, pressures in the biliary system rise to levels well above pancreatic secretory pressures and reflux of bile up the pancreatic duct can then occur. Recent work by Elliott[3] has also shown that bile incubated 24 hours with pancreatic juice (theoretically in the gall bladder when obstruction has created a common channel) is altered so that it is accepted by the pancreatic duct system at pressures within the physiologic range.

Thus, in the 30 to 70 per cent of persons who have the anatomic possibility of a common channel, biliary reflux appears to be a significant factor in the etiology of pancreatitis. In those remaining persons, whose common bile duct and pancreatic duct open side by side at the papilla of Vater or open separately into the duodenum, other etiological factors must be sought in explanation of the pancreatitis. The factor which has appeared most reasonable and attractive is that of pancreatic duct obstruction.

Many authors have, indeed, claimed duct obstruction to be the basic etiological factor in all cases of pancreatitis. Bell[1] has summed up this viewpoint recently when he wrote, "It is well established that pancreatitis is due to rupture of the small pancreatic ducts with escape of pancreatic secretion into the parenchyma of the gland. The rupture is caused by increased intraductal pressure which is brought about by increased pancreatic secretion associated with some degree of obstruction to its outflow."

This factor was originally given extensive study by Rich and Duff[12] in their critique of 1936. They found partial duct obstruction in 13 of their 24 cases and it was their opinion that this was the basic etiological factor in most cases of pancreatitis. More recently, Thal[16] has reported evidence of ductal obstruction in the "vast majority" of his study of 42 fatal cases. He further found that 80 per cent of the patients studied had evidence of recurring acute exacerbations of chronic pancreatic disease extending over periods of months to years. Thus, duct obstruction plus episodic recurrences appears to be significant.

The main objection to duct obstruction as a basic etiological factor in pancreatitis has been that in the experimental studies, in general, an edematous pancreatitis only has been produced. In these experimental studies of duct obstruction, only one type of obstruction has been thoroughly studied—complete obstruction. Popper,[10] in dogs, and Lium[5] and Wangensteen,[18] in cats, found that an edematous pancreatitis occurs after ligation of the duct and stimulation of the gland. Following this initial edematous response and mild inflammation, the pancreas becomes atrophied. Duct ligation and stimulation of the gland has become the classic method of creating an edematous and moderately severe pancreatitis. Chronic pancreatitis has not been reported from duct obstruction and acute necrotizing or hemorrhagic pancreatitis has never been created in dogs by this method alone.

Popper[11] has shown, however, that ischemia of the pancreas for a relatively brief span of time will convert this edematous pancreatitis to a hemorrhagic pancreatitis. These studies utilized duct ligation, stimulation of the gland with intravenous secretin, and clamping of the pancreatic artery for 15 minutes. Palmer[9] has stated that vascular injury is the "common denominator" in

hemorrhagic pancreatitis. Rich and Duff[12] have ascribed the vascular injury to the action of the activated pancreatic enzymes in the interstices of the gland. These active enzymes in contact with the pancreatic vessels cause the thrombosis and vascular necrosis so commonly seen.

This concept, while accepted by most, has not been demonstrated experimentally with duct obstruction and gland stimulation alone. The interstitial fluid of edematous pancreatitis contains the inactive enzyme precursors trypsinogen and chymotrypsinogen. These must be activated or vascular ischemia must be added to create a hemorrhagic pancreatitis. Stein[15] has activated the enzyme by the injection of intra-arterial trypsin with resultant hemorrhagic pancreatitis. It is felt by many that the reflux of bile into the pancreas creates hemorrhagic pancreatitis by the activation of these enzymes. Others have attempted to create vascular ischemia and injury by injecting finite particles into the pancreatic artery to occlude it.[14]

In spite of these many studies of complete ligation of the pancreatic ducts, we have found no reported studies on partial duct obstruction and only one previous study of intermittent duct obstruction.[17]

The characteristic response of secretory organs to complete obstruction is one of early distention and inflammation, succeeded by atrophy of the organ. After incomplete obstruction, however, so long as some secretion is possible, atrophy does not occur. Distention and stasis with repeated episodes of inflammation occur and the resistance of the organ to infection becomes decreased. Essentially, the element of time has been added to the factor of obstruction.

Clinically, it would appear that when obstruction of the pancreatic duct system occurs, it is most frequently partial or intermittent. Intermittent duct obstruction is usually caused by edema and spasm of the duodenum and/or sphincter.[4] It also occurs from reflex mechanisms, from local inflammation as in alcoholic duodenitis, or from biliary distention and pain. The obstruction caused by ductal metaplasia, considered important by Rich and Duff, is a partial obstruction. Fibrosis of the sphincter creates a partial duct obstruction. Common duct stones, associated with about 5 per cent of pancreatitis, usually block the pancreatic duct incompletely. Indeed, when complete pancreatic duct obstruction occurs in the human being, as in surgical ligature of the pancreatic duct, pancreatitis is unusual.

In the conditions listed above, chronic incomplete duct obstruction is present. Because of the frequent association of this condition with pancreatitis, we believe that a study of the experimental effects of incomplete pancreatic duct obstruction with episodic pancreatic stimulation over a period of time should be undertaken.

This study evaluates the effect of partial and intermittent pancreatic duct obstruction over a period of several weeks, attempting to condense by more frequent episodes of stimulation and obstruction what might occur clinically over a period of months to years.

METHODS

All studies were made with mongrel dogs weighing 10 to 15 kilograms. Dogs have their major pancreatic drainage via the accessory pancreatic duct. This duct enters the

duodenum separately without joining the common bile duct. The smaller pancreatic duct in the dog, which is very small, joins the common bile duct. In this study we have directed our attention solely to the major pancreatic drainage, the accessory pancreatic duct, because previous studies have shown us that the lesser duct is important only in pressure studies or when the major duct is completely obstructed. Anesthesia was obtained by use of intravenous sodium pentothal (Nembutal), 30 mg. per kilogram of body weight.

Partial Duct Obstruction.—Laparotomy was performed in 6 dogs with exposure of the pancreas and partial ligation of the major pancreatic duct over a No. 24 needle. This narrowed the duct to about one third of its normal internal diameter. The abdomen was then closed and the dogs allowed to recover.

Following this partial duct obstruction, the pancreas was stimulated every third to fourth day. Stimulation was effected by a heavy protein and fat meal of meat and milk (1½ cans of high protein dog food with one-half quart of milk) and by injecting subcutaneously at the same time 10 U. of secretin* and 0.75 mg. of Urecholine. To ensure an especially hungry dog on the days of stimulus, the dog was not fed the previous day. Blood amylase values were determined preoperatively and at varying intervals thereafter. The dogs were serially sacrificed at 5, 10, 15, 20, 30, and 40 days after the partial obstruction and the pancreas was examined grossly and histologically.

Intermittent Duct Obstruction.—Exploratory operations were performed on 10 dogs with exposure of the pancreas and the placement of a "choker" around the major pancreatic duct. This choker consisted of a small polyvinyl catheter (internal diameter, 0.044 cm.) threaded over a No. 7 stainless steel wire to give added strength. This was passed beneath the duct and looped so that both ends then passed through a larger polyvinyl catheter which acted as the occluding choker. The duodenal loop was then sutured to the anterior abdominal wall and the choker was brought out through the abdominal fascia, which was closed about it. It was buried in the subcutaneous tissue so that removal of 3 to 4 skin sutures exposed it to manipulation. The dogs were allowed to recover.

On the days of laparotomy and at standard intervals thereafter (see below) the choker was tightened to occlude the pancreatic duct and the pancreas was stimulated to secrete. On these days the dog was lightly anesthetized, the skin incision opened, and the choker tightened. Since the dogs were asleep at the time of obstruction, pancreatic stimulation was obtained by intravenous drip of secretin, 50 U. per hour, and Urecholine, 1.25 mg. per hour, in 5 per cent dextrose in saline solution.

The ducts in 2 dogs were obstructed for 3 hours and the pancreas stimulated for 2 hours, every fourth to fifth day, for a total of 5 obstructive periods in 3 weeks. The dogs were then sacrificed.

In 2 dogs the time intervals between obstruction-stimulation episodes were shortened. The ducts in these dogs were obstructed for 3 hours and the pancreas was stimulated for 2 hours every second day, for a total of 6 episodes in 2 weeks. The dogs were then sacrificed.

The ducts in the remaining 6 dogs were obstructed for 4 hours and the pancreas stimulated for 3 hours with a time interval of 4 days between episodes. This represents an increase of 1 hour in the obstruction-stimulation period. Of these 6 dogs, 4 died within 1 week and 2 were sacrificed within the week because of their seriously ill state.

Blood amylase values were obtained in all dogs at varying intervals after the periods of obstruction-stimulation. The pancreas was taken for gross and microscopic study at the time of sacrifice or death.

RESULTS

Amylase Values.—Amylase determinations were reported in Meyer units.[8] This unit equals about one fifth of the Somogyi unit. Preoperatively and in normal dogs we have found amylases values in the range of 100 to 300 U. (from 4 to 10 times the normal in the human being). The normal dog amylase varies

*Courtesy Eli Lilly Company, Indianapolis, Ind.

widely and is elevated in many conditions. That much of the circulating amylase is not from a pancreatic source is shown by a level of 100 U. which we found in 1 dog 10 days after total pancreatectomy.

Fig. 1 illustrates the range of amylase values found in the 6 dogs with partial duct obstructions. These values were drawn at varying intervals as shown. It is noted that all values (except one) obtained in the first 2 weeks after partial ligation were elevated above the normal range with a wide distribution in the values obtained. There is a decrease in the values after the first 10 days.

Fig. 2 shows the values obtained in the 10 dogs with intermittent pancreatic duct obstruction. In these dogs the initial rise is much more pronounced with values 100 per cent greater than the normals. Again, there is a decline in values over the next 2 weeks.

The initial higher amylase values may, in part, be due to operative handling of the duodenum and pancreas during exposure of the pancreatic duct at laparotomy. The effect of the manipulation would be existent only 24 to 36 hours.

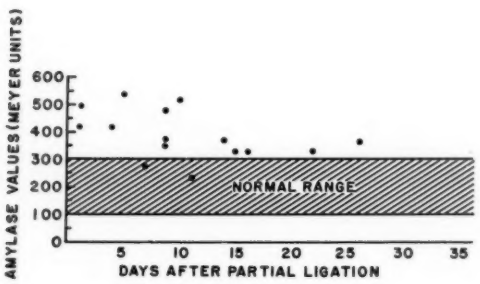


Fig. 1.

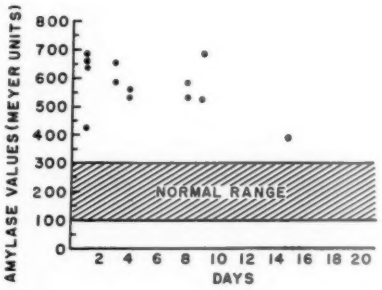


Fig. 2.

Fig. 1.—Amylase values after partial pancreatic duct obstruction.
Fig. 2.—Amylase values with intermittent duct obstructions (Day 0 is day of initial obstruction).

That the amylase levels in the dogs with intermittent duct obstructions were higher than those with partial duct obstructions probably reflects two mechanisms. First, a more complete obstruction is imposed during the period of pancreatic secretion. Second, we believe it evident from the results, that a greater stimulus to secrete was obtained by the intravenous solutions than was obtained by the meat and milk feedings and subcutaneous injection. The level of blood amylase apparently rises in proportion to the amount of pancreatic stimulation and the completeness of obstruction in the duct system. It does not reflect with accuracy the severity of the pancreatitis, as noted in many clinical reports.[13] The amylase values obtained in the 4 dogs which died within 1 week were within the range of the others in the group with intermittent duct obstructions.

Course of Observation.—All dogs with partial duct obstruction survived the time intervals to be sacrificed. They exhibited mild degrees of illness only with lethargy and lack of appetite as the prominent symptoms.

In the group of dogs with intermittent duct obstruction, the 4 with obstruction for 3 hours and stimulation for 2 hours lived to be sacrificed at 2 and 3 weeks. They displayed moderate degrees of illness such as anorexia, lethargy, some nausea, diarrhea, and occasional vomiting. The addition, in 6 dogs, of 1 hour of obstruction and stimulation for a total of 4 hours of obstruction and 3 of stimulation appeared to be lethal. It caused the death of 4 dogs. The other 2 dogs were severely ill and were sacrificed at the end of 1 week. Two dogs died within 72 hours of the first episode and 2 dogs died within 48 hours of the second episode of obstruction and stimulation. These 6 dogs had severe anorexia and nausea, frequently vomited, and had a watery-brown diarrhea.

No dog had evidence of pancreatic insufficiency by stool inspection. No antibiotics or intravenous therapy was given except for the solutions given on the days of stimulation to the group of dogs with intermittent obstruction.

Gross and Microscopic Findings.—In the dogs with partial duct obstruction, the glands varied on gross inspection from a moderately boggy and indurated gland in the dog sacrificed at 5 days to a firm, nodular, and hard gland in the dog sacrificed at 40 days. There was no gross evidence of fat necrosis or hemorrhage in the glands. There was no serosanguineous fluid in the abdominal cavity. All ducts were examined and found to be patent with stenosis at the point of partial ligature and varying degrees of dilatation of the duct behind this point.

Microscopic sections of these 6 glands revealed the following conditions: In the dog sacrificed at 5 days, there was classical edematous pancreatitis with interlobular and interstitial edema, mild round cell infiltration, early organization, and fibroblastic proliferation (Fig. 3). In the glands of dogs sacrificed at 10, 15, 20, and 30 days, the amount of edema seen was decreased, the round cell infiltration was increased, the organization and fibroplasia was increased. There was pronounced periductal and interlobular fibrosis in the dogs sacrificed after 2 weeks (Fig. 4). In the pancreas of the dog sacrificed at 40 days, there was marked fibrosis and scarring throughout, especially in the periductal and interlobular regions, with many areas in which the acini have been replaced with scar tissue (Fig. 5). This gland shows in many areas, the changes considered typical of chronic pancreatitis.

In the dogs with intermittent duct obstruction, the 4 dogs with obstruction for 3 hours and stimulation for 2 hours, which were sacrificed at 2 and 3 weeks, showed changes in the pancreas similar to those just mentioned. On gross inspection, these glands were nodular, indurated, and slightly contracted. There was no fat necrosis or hemorrhage seen grossly. No serosanguineous fluid was found in the peritoneal cavity. The ductal system was only slightly dilated behind the point of intermittent obstruction and the ducts were patent.

Microscopic sections of these glands revealed much interlobular and periductal fibrosis with round cell and polymorphonuclear infiltration. A prominent feature was marked vascular congestion throughout the pancreas. No areas of hemorrhage were seen (Fig. 6). In one dog, one focal area of necrosis was seen. Fat necrosis was seen microscopically surrounding all the glands in this group.

Fig. 3.

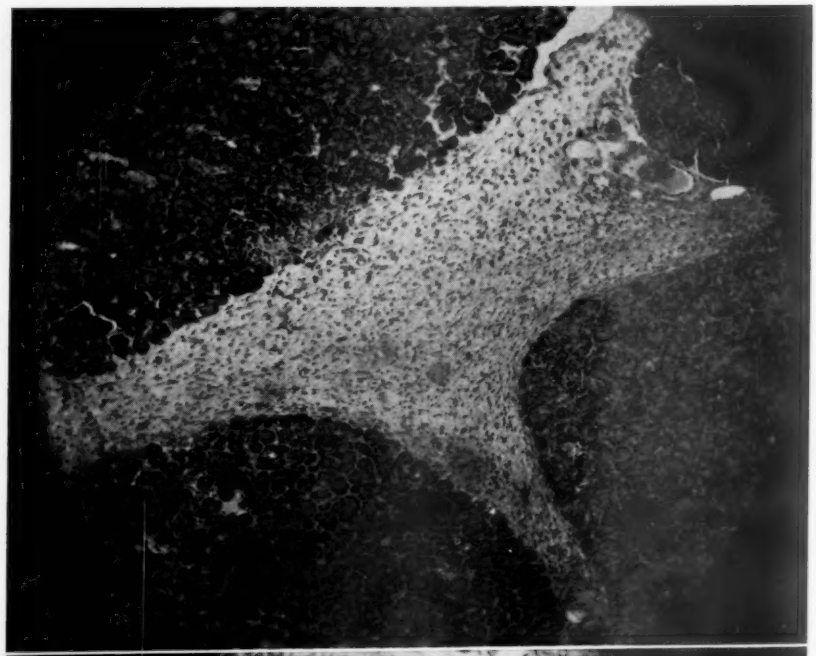


Fig. 4.

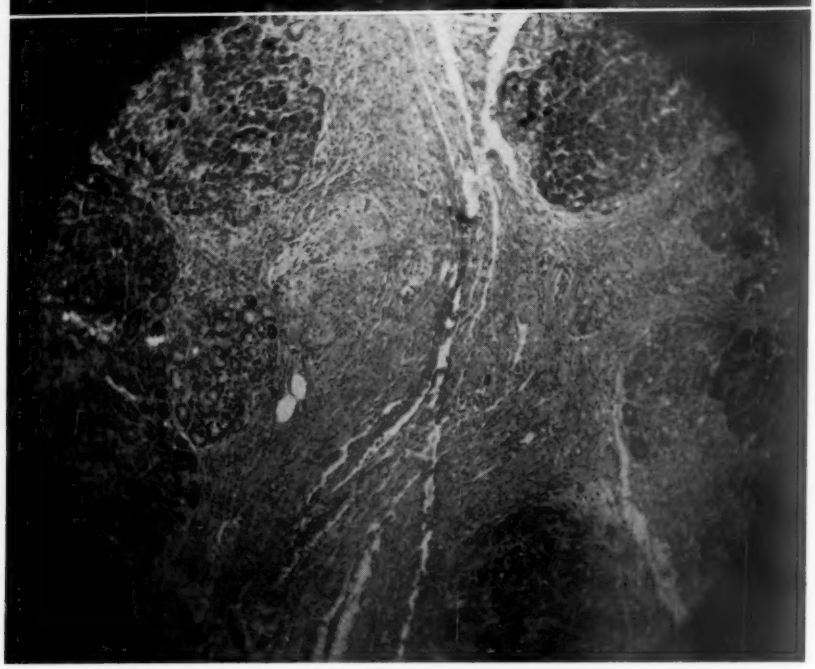
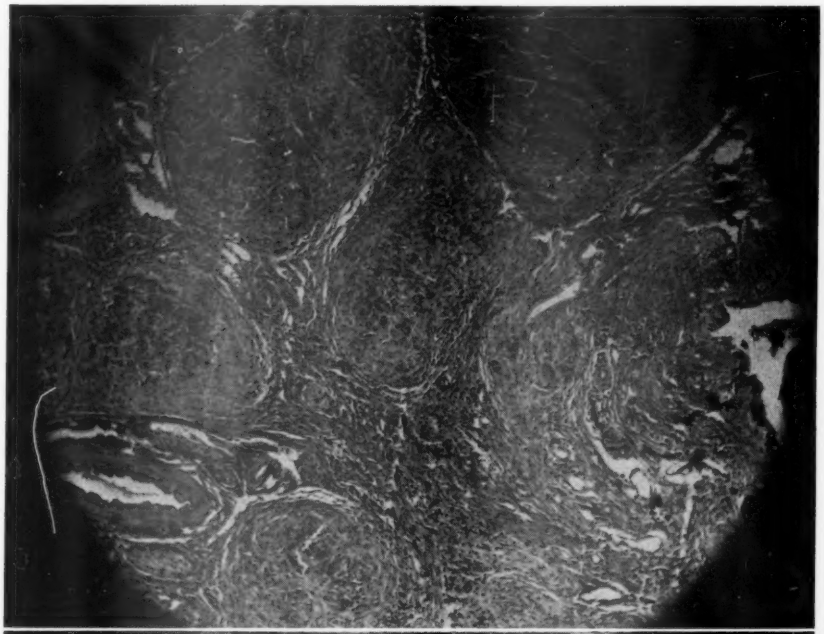


Fig. 3.—Edematous pancreatitis. Microscopic section (×10) of pancreas of dog sacrificed at 5 days. There is marked interlobular edema with round cell infiltration, early organization, and fibroplasia.

Fig. 4.—Edematous pancreatitis with chronic changes (×10). Interlobular edema persists with increased round cell inflammation. Organization and fibroblastic proliferation is increasing with interlobular and periductal fibrosis becoming pronounced.

Fig. 5.



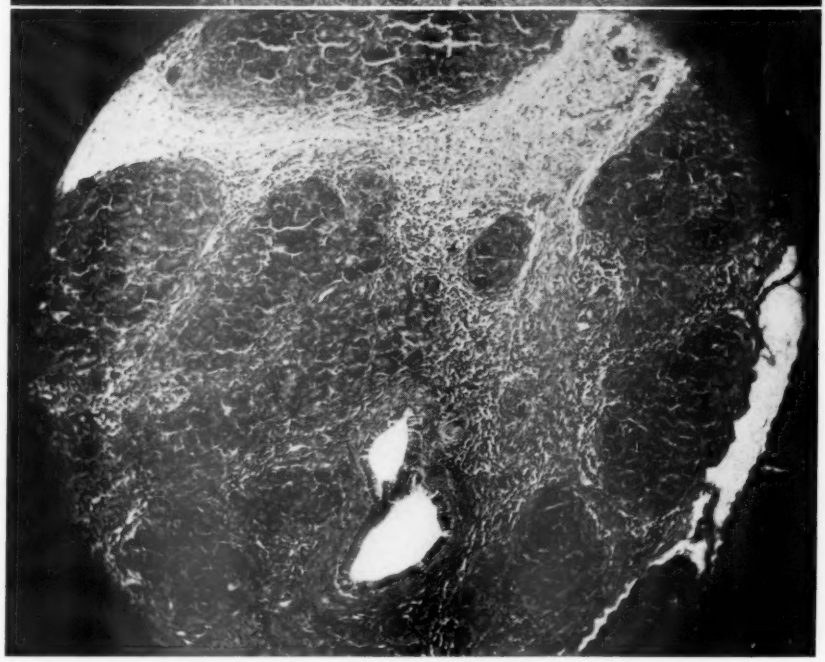


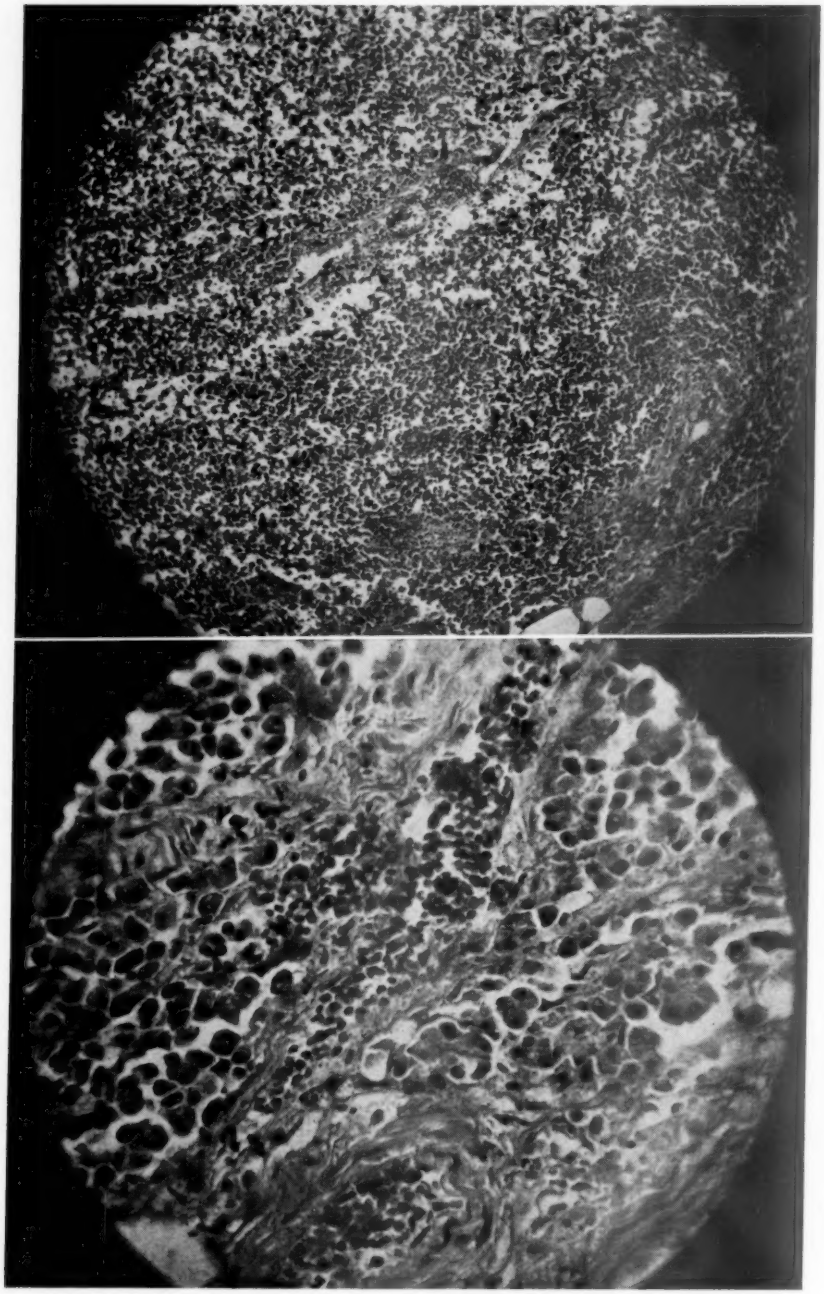
Fig. 6.

Fig. 5.—Chronic pancreatitis (×10). Dog sacrificed at 40 days. There is marked fibrosis and scarring, most evident in the interlobular and periductal areas. Many lobules are completely replaced by fibrosis.

Fig. 6.—Edematous pancreatitis with chronic changes (×10). Interlobular edema with marked round cell inflammation, vascular congestion, early interlobular and periductal fibrosis.

In those 6 dogs with a more lethal amount of obstruction and stimulation, 4 and 3 hours, respectively, a somewhat different picture was seen. In the 4 dogs that died, postmortem examination was not procured until 4 to 8 hours after death since they died sometime during the night. Early postmortem

A.



B.

Fig. 7.—*A*, Hemorrhagic pancreatitis (×10). Section of area of focal necrosis with areas of hemorrhage. The inflammation is both round cell and polymorphonuclear in type. *B*, Hemorrhagic pancreatitis (×43). High-power view showing vascular congestion with dissolution of vessel wall and hemorrhage.

changes were seen, both grossly and microscopically, and had to be separated from the antemortem changes. For this reason, the remaining 2 dogs were sacrificed short of death when they appeared severely ill. It was our opinion that these 2 dogs would have eventually died if allowed to go on to termination.

Gross examination of the pancreas of the dogs which died revealed a more boggy indurated gland. Grossly, there were areas of hemorrhage and some areas of fat necrosis. There was approximately 50 ml. of serosanguineous effusion in the peritoneal cavity at the time of autopsy. The marginal pancreaticoduodenal vein was found to be thrombosed in these animals.

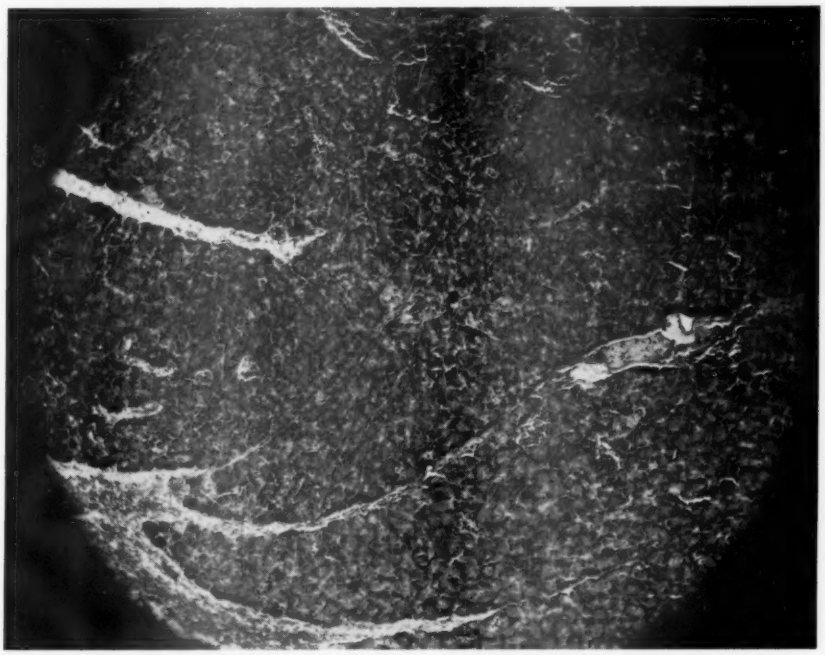


Fig. 8.—Hemorrhagic pancreatitis (×10). Area showing prominent vascular congestion in lobules and interlobular hemorrhage *(left lower corner)*.

Microscopic sections of these glands revealed autolytic postmortem changes in many areas. Antemortem changes seen were fat necrosis around the gland, multiple focal areas of hemorrhage with marked vascular congestion of the intrapancreatic vessels, many thrombosed vessels, with a few focal areas of necrosis (Fig. 7). There was also evidence of fibrosis and fibroblastic proliferation in the 2 dogs that lived through one obstruction-stimulation period 4 days previously.

In the 2 dogs sacrificed in a severely ill state, gross examination of the pancreas showed it to be boggy, congested, and indurated. There was marked congestion of the pancreatic vessels. No gross fat necrosis was visible. There was no fluid in the peritoneal cavity. Microscopic sections of these glands again revealed a striking amount of vascular congestion, many with evidence of rupture and hemorrhage into the pancreas. No thrombosed vessels were seen (Fig. 8). There were no areas of necrosis except for fat necrosis about the gland. Infiltration in the interlobular areas was predominantly of a round cell type.

SUMMARY AND CONCLUSIONS

The objection to the common channel theory as the primary etiological factor in pancreatitis has been that only 30 to 70 per cent of persons have the anatomic configuration of the ducts to make such a theory tenable. For this reason, other etiological factors have been sought to explain those cases in which an anatomic common channel does not exist. The factor of pancreatic duct obstruction has long appeared attractive to many. However, experimental efforts to produce all degrees of pancreatitis with complete duct obstruction have not been successful. One form only, that of edematous pancreatitis, has in general been consistently produced. Chronic pancreatitis has especially not been produced because of the atrophy of the gland which occurs as a sequel to complete ligation. Hemorrhagic pancreatitis is produced only with the addition of other factors.

We have studied incomplete pancreatic duct obstruction, utilizing the 2 mechanisms of partial ligation of the duct and intermittent duct obstruction. By intermittent stimulation of the pancreas to secrete, we have attempted to reproduce the episodic recurrences characteristic of the clinical course of the majority of patients with pancreatitis who are studied at postmortem examination.

In this study, as well as attempting to evaluate the effects of these 2 types of incomplete duct obstruction, we have evaluated variable stimuli and varying time intervals between stimuli. We have found the most lethal results to be associated with intermittent pancreatic duct obstruction of 4 hours' length with intravenous stimulation of the pancreas of 3 hours' length.

We have created, in the 16 dogs studied, the full range of inflammatory diseases of the pancreas. The dogs sacrificed early showed edematous pancreatitis. Those which survived periods of 2 weeks to 40 days (the longest period studied) revealed successive degrees of chronic pancreatitis. Of the 6 dogs obstructed 4 hours and stimulated 3 hours, 4 dogs died. The other 2 dogs in this group were severely ill and were sacrificed prior to death. Histologic study of the pancreas from these dogs revealed severe congestion of the pancreatic vessels with many areas of hemorrhage and areas of focal necrosis. There were thrombi in the vessels of those dogs that died. These findings are those of hemorrhagic pancreatitis.

Many authors have emphasized the importance of a vascular factor in the etiology of hemorrhagic pancreatitis. This factor has been previously added by vascular ischemia or injury. No prior demonstration of the production of vascular congestion with thrombosis and hemorrhage from duct obstruction and pancreatic stimulation alone has been reported. We believe our results support the thesis of vascular injury from congestion in the etiology of hemorrhagic pancreatitis.

With incomplete pancreatic duct obstruction and episodic stimulation of pancreatic secretion, all degrees of pancreatitis are possible. With mild stimulation, early in the course of the disease, edematous pancreatitis occurs. Over a prolonged period, with further episodes, chronic pancreatitis ensues. Finally,

with near maximal stimulation of the gland to secrete, in the presence of obstruction to this secretion, vascular congestion and injury occurs with hemorrhage, areas of focal necrosis, and a fatal termination.

REFERENCES

1. Bell, E. T.: Pancreatitis, SURGERY **43**: 527, 1958.
2. Colp, R., and Dreiling, D. A.: The Pancreas *in* Zimmerman and Levine, Physiologic Principles of Surgery, Philadelphia, 1957, W. B. Saunders Company, p. 665.
3. Elliott, D. W., Williams, R. D., and Zollinger, R. M.: Alterations in the Pancreatic Resistance to Bile in the Pathogenesis of Acute Pancreatitis, Ann. Surg. **146**: 669, 1957.
4. Ivy, A. C.: Pancreatitis: A Review, SURGERY **31**: 614, 1952.
5. Lium, R., and Maddock, S.: Etiology of Acute Pancreatitis, SURGERY **24**: 593, 1948.
6. Mann, F. C., and Giordano, A. S.: The Bile Factor in Pancreatitis, Arch. Surg. **6**: 1, 1923.
7. Menguy, R. B., Hallenbeck, G. A., Bollman, J. L., and Grindlay, J. H.: Intraductal Pressures and Sphincteric Resistance in Canine Pancreatic and Biliary Ducts After Various Stimuli, Surg. Gynec. & Obst. **106**: 306, 1958.
8. Meyer, V. C., Free, A. H., and Rosinski, E. R.: Studies on Animal Diastases. VI. The Determination of Diastase (Amylase) in Blood, J. Biol. Chem. **154**: 39, 1944.
9. Palmer, E. D.: Clinical Gastroenterology, New York, 1957, Paul B. Hoeber, Inc., p. 574.
10. Popper, H. C., and Necheles, N.: Edema of the Pancreas, Surg. Gynec. & Obst. **74**: 123, 1942.
11. Popper, H. L., Necheles, H., and Russell, K. C.: Transition of Pancreatic Edema Into Pancreatic Necrosis, Surg. Gynec. & Obst. **87**: 79, 1948.
12. Rich, A. R., and Duff, G. L.: Experimental and Pathological Studies on the Pathogenesis of Acute Pancreatitis, Bull. Johns Hopkins Hosp. **58**: 213, 1936.
13. Richman, A.: Acute Pancreatitis, Am. J. Med. **21**: 246, 1956.
14. Smyth, C. J.: Etiology of Acute Hemorrhagic Pancreatitis With Special Reference to the Vascular Factors, Arch. Path. **30**: 651, 1940.
15. Stein, A. A., Powers, S. R., and Browns, H. H.: Experimental Hemorrhagic Pancreatitis, New Concepts of Pathogenesis, Ann. Surg. **143**: 508, 1956.
16. Thal, A. P., Perry, J. F., and Egner, W.: A Clinical and Morphologic Study of Forty-Two Cases of Fatal Acute Pancreatitis, Surg. Gynec. & Obst. **105**: 191, 1957.
17. Waite, J. H.: Recurring Pancreatitis in Dogs, Proc. Surg. Forum, Am. Coll. Surgeons, Philadelphia, 1952, W. B. Saunders Company, p. 516.
18. Wangensteen, O. H., Leven, N. L., and Manson, M. H.: Acute Pancreatitis: An Experimental and Clinical Study With Special Reference to the Significance of the Biliary Tract Factor, Arch. Surg. **23**: 47, 1931.

THE USE OF AN EXTERNAL BYPASS DURING EXPERIMENTAL TOTAL HEPATECTOMY

HARRY A. KAUPP, JR., M.D., AND THOMAS E. STARZL, M.D., PH.D.,*
CHICAGO, ILL.

(From the Departments of Surgery, Northwestern University Medical School and U. S. Veterans Administration Research Hospital)

RECENTLY, a new method was described for complete one-stage hepatectomy in dogs.[1] The technique involved preliminary preparation of a side-to-side portacaval shunt and ligation of the portal triad. The inferior vena cava was then temporarily occluded above and below the liver, and the liver removed in a bloodless field by individual ligation of the hepatic veins with preservation of the inferior vena cava. During the 15 or 20 minutes of actual hepatectomy, the confluent (via the portacaval anastomosis) portal and inferior vena caval flow was blocked with no apparent permanent harm to the bowel. With release of the temporarily occluding caval clamps, free venous return of both the splanchnic and inferior vena caval drainage was immediately restored.

Since the publication of this technique, several investigators without formal surgical training have attempted to apply this method to their investigations at this institution. Their time for removal of the liver during portacaval occlusion has greatly exceeded the 15 or 20 minutes described by us. Some of their animals have developed hypotension or hemorrhagic gastroenteritis secondary to the prolonged portacaval hypertension. In order to avoid these complications and to make this experimental method useful to a larger number of investigators, an external by-pass has been developed which can be used during the period of liver removal to avoid the immediate and late effects of prolonged acute portal hypertension.

METHOD

Mongrel dogs of 20 to 40 kilograms are employed. The operation is performed essentially as previously reported.[1] After completion of the portacaval anastomosis, ligation of the portal triad, and mobilization of the liver, cut-downs are performed over the femoral vein in the femoral triangle and the external jugular vein in the neck. A ¼ inch (inside diameter) polyethylene catheter which has been siliconized† is inserted into the inferior vena cava through the femoral vein, led alongside the dog and inserted into the neck vein (Fig. 1). Temporary occlusion of the vena cava is immediately carried out, and the liver removed as previously described.[1] A brisk flow of blood from

Aided by Grant A-3176 from the United States Public Health Service, National Institutes of Health, Bethesda, Md.

Received for publication Jan. 5, 1960.

*Markle Scholar.

†Compound Z-4141, Dow Corning Corporation, Midland, Michigan.

the inferior to the superior vena cava occurs through the external shunt as long as the portacaval pressure remains high or until clots form in the polyethylene catheter. If the period of portacaval occlusion is unduly prolonged, clots will form in the external shunt after 30 to 45 minutes. As soon as the liver is out and the vena cava released, the external shunt is removed, and the femoral and external jugular veins ligated. The duration of the operation is increased by no more than 5 or 10 minutes by the placement and removal of the external shunt.

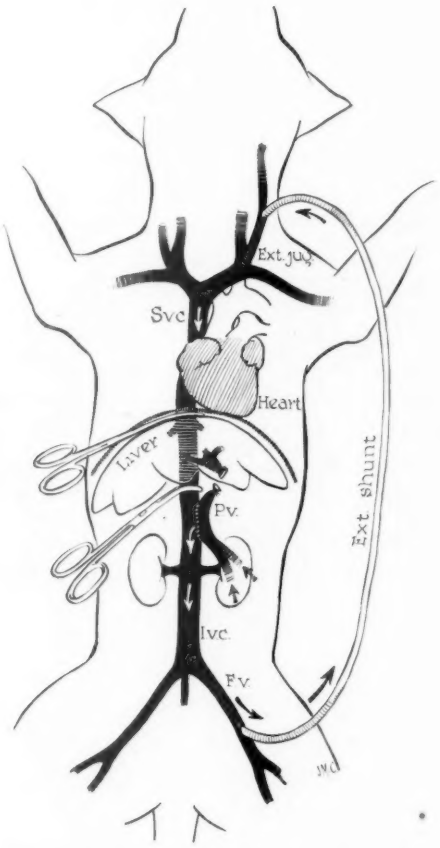


Fig. 1.—Polyethylene shunt from femoral to external jugular vein, which is used during the period of liver removal to decompress the portal and caval systems.

SUMMARY

An external bypass has been developed which is designed to prevent acute portacaval hypertension during the performance of a recently described method of hepatectomy. The temporary external shunt has considerably prolonged the safe period of portacaval occlusion, and extended the use of this method of hepatectomy to personnel relatively untrained in surgery.

REFERENCES

1. Starzl, T. E., Bernhard, V. M., Benvenuto, R., and Cortes, N.: A New Method for One-Stage Hepatectomy for Dogs, SURGERY **46**: 880, 1959.

THE SURGICAL MANAGEMENT OF MASSIVE UPPER GASTROINTESTINAL HEMORRHAGE DUE TO CARDIOESOPHAGEAL MUCOSAL LACERATIONS: THE MALLORY-WEISS SYNDROME

L. H. Stahlgren, M.D., and C. S. Ling, M.D., Philadelphia, Pa.

(From the Philadelphia General Hospital)

THE surgical management of patients with massive upper gastrointestinal hemorrhage due to the Mallory-Weiss syndrome has received less attention than it deserves.[4, 5] Mallory and Weiss[3] first called attention, in 1929, to the syndrome which bears their names. They reported autopsy findings on 4 patients who died from gastrointestinal bleeding arising from lacerations of the cardioesophageal mucosa. The lacerations extended in a linear fashion from the gastric cardia toward and, in approximately one-half the patients, into the distal esophagus. The 4 patients reported a history of prolonged vomiting associated with alcoholism. Mallory and Weiss attributed the mucosal lacerations to trauma from retching. The retching caused forceful retrograde propulsion of gastric contents against the insufficiently dilated cardia and esophagus. The two doctors reproduced such conditions in postmortem stomachs by obstructing the pylorus and exerting pressure on the gastric contents toward the partially constricted lower esophagus.

Factors which have been commonly associated are alcoholic gastritis, atrophic gastritis, and nutritional deficiencies, all of which may have predisposed the mucosa to injury.[2, 4, 5]

Mallory and co-authors have reported 17 patients in whom they had confirmed this diagnosis at autopsy over a 25-year period.[2, 3, 7] On the basis of their experience with this syndrome, they recommend that the surgeon examine the mucosal folds in the gastric cardia in all patients who are operated upon for upper gastrointestinal bleeding of obscure origin. Three of the patients in their series underwent exploratory operation before death but in each instance the surgeon failed to identify the bleeding site.

Decker, Zamcheck, and Mallory have pointed out the risk of performing "blind" gastrectomies in such patients.[2] While we favor blind gastric resection in certain circumstances,[1] we now recommend a preliminary gastrotomy and thorough inspection of the cardioesophageal area in patients with a history of alcoholism or those in whom blood-free vomiting or retching preceded hematemesis. The patient in our report was managed in this manner.

CASE REPORT

Case Report.—E. W., a 50-year-old Negro man, was admitted to the Philadelphia General Hospital on Feb. 12, 1959, because of hematemesis which had begun 6 hours prior to admission. The hematemesis had been preceded by nausea and daily vomiting for a

Received for publication Feb. 5, 1960.

period of 2 weeks. The patient's family reported that he was an alcoholic and had been consuming considerable quantities of alcohol daily. During an admission 3 years before for a hemorrhoidectomy, an upper gastrointestinal barium x-ray was normal. There was a past history which was suggestive of peptic ulcer.

On examination, the patient was pale, blood pressure was 90/70 mm. Hg, pulse rate was 90 per minute and the respiratory rate was 20 per minute. There were no stigmas of cirrhosis. There was tenderness in the right upper quandrant and midepigastrium.

Liver function studies were normal, the prothrombin time was 23 seconds, and the hemoglobin was 13.3 Gm. The bleeding and coagulation times were normal.

Treatment consisted of whole blood replacement and an ulcer diet. Because the diagnosis of esophageal varices could not be excluded, a Sengstaken-Blakemore tube was inserted as a diagnostic test. Esophageal tamponade momentarily stopped the bleeding, but it promptly recurred. During the 12 hours following admission, the patient vomited 7 times with an estimated total blood loss of 5 L. Despite transfusion of 8 pints of whole blood in less than 12 hours, the hemoglobin dropped from 13.3 Gm. at time of admission to 8.2 Gm. just prior to operation. The pulse rate rose from 90 to 120 per minute.

Operation: At laparotomy, the stomach was filled with blood but showed no evidence of ulceration, scarring, or tumor. The duodenum was normal. An incision was placed in the prepyloric area and the blood clots evacuated. Blood was observed trickling down from the cardiac end of the stomach. The gastrotomy was then extended upward to the midstomach and the mucosal folds near the esophagogastric junction were inspected. Two fissurelike lacerations on the posterior lateral surface of the stomach were seen. One extended approximately 0.5 cm. into the esophagus. With downward traction on the posterior wall of the stomach, the cardia was exposed and a suture of fine atraumatic catgut used to oversew the most proximal laceration. With tension on the suture, the higher laceration was brought into view and oversewn in a similar manner. This stopped the bleeding. The patient recovered uneventfully and was discharged from the hospital.

Comment: This history of upper gastrointestinal bleeding beginning after an alcoholic debauch associated with repeated vomiting is suggestive of the Mallory-Weiss syndrome. Of interest is the fact that esophagogastric tamponade failed to arrest the hemorrhage. It is likely that this patient would have become exsanguinated without surgical control of the bleeding point.

DISCUSSION

Two undesirable results may follow a surgeon's failure to appreciate the significance of cardioesophageal mucosal lacerations as a cause of massive upper gastrointestinal hemorrhage. The bleeding point may be overlooked at operation if the rugal folds in the gastric cardia are not carefully inspected. In that event, recurrent or persistent hemorrhage may be expected.

Furthermore, the patient may be subjected to an unnecessary subtotal gastric resection or, as recently reported, a total gastrectomy.[6] The experience we gained from the case described above has caused us to modify our concept of the operative management of patients who are bleeding massively from an obscure upper gastrointestinal lesion. We now believe that a gastrotomy with inspection of the cardiac end of the stomach should be considered in such cases. This procedure is recommended in patients in whom the bleeding was preceded by a period of vomiting or retching, or in those in whom there was a recent bout of alcoholism. At operation, the gastrointestinal tract should be carefully inspected with particular emphasis on investigation of the stomach and duodenum. If the source of bleeding is not found, a gastrotomy is performed in

the midstomach. The surgeon may be able to identify one or more shallow lacerations extending toward the esophagus, near the esophagogastric junction. Downward tension on the posterior wall of the stomach will bring the cardia near the gastrotomy incision. The bleeding point can then be ligated. It is unnecessary to mobilize and incise the esophagus unless the laceration extends a considerable distance into that structure. Tension on the suture will deliver the esophageal laceration into view.

SUMMARY AND CONCLUSIONS

The Mallory-Weiss syndrome should be ruled out in any patient with massive upper gastrointestinal hemorrhage preceded by retching in whom the bleeding lesion cannot be found at operation. In this syndrome, the mucosal laceration is located near the esophagogastric junction, and can be identified by a gastrotomy through which downward traction is exerted on the posterior wall of the stomach.

Once the diagnosis of cardioesophageal mucosal laceration is established, the bleeding can be controlled by suture of the lacerations. Therefore, it is imperative that the surgeon should not only correctly identify the source of bleeding but that he should also avoid a major procedure such as a gastric resection in an effort to control it.

REFERENCES

1. Cooper, D. R., Stahlgren, L. H., Sylvester, L. E., and Ferguson, L. K.: Surgical Treatment of Massive Upper Gastrointestinal Hemorrhage, Gastroenterology **34**: 947, June, 1958.
2. Decker, J. P., Zamcheck, N., and Mallory, G. K.: Mallory-Weiss Syndrome. Hemorrhage From Gastroesophageal Lacerations at the Cardiac Orifice of the Stomach, New England J. Med. **249**: 957, 1953.
3. Mallory, G. K., and Weiss, S.: Hemorrhages From Lacerations of the Cardiac Orifice of the Stomach Due to Vomiting, Am. J. M. Sc. **178**: 506, 1929.
4. McPhedran, N. I.: Massive Upper Gastrointestinal Bleeding From Spontaneous Laceration of the Lower Esophagus (Mallory-Weiss Syndrome), Canad. J. Surg. **2**: 103, 1958.
5. Small, A. B., and Ellis, P. R.: Laceration of the Distal Esophagus Due to Vomiting (The Mallory-Weiss Syndrome), New England J. Med. **258**: 285, 1958.
6. Shuttleworth, K. E. D., and Hutt, M. S. R.: Mallory-Weiss Syndrome; A Case With Recovery After Total Gastrectomy, Brit. J. Surg. **46**: 1, 1958.
7. Weiss, S., and Mallory, G. K.: Lesions of the Cardiac Orifice of the Stomach Produced by Vomiting, J. A. M. A. **98**: 1353, 1932.

TRACHEAL DIVERTICULA

Report of a Specimen and Review of the Literature

C. R. Dutta, M.B.B.S., and J. V. Basmajian, M.D., Kingston, Ontario

(From the Department of Anatomy, Queen's University)

DURING routine dissection of a male cadaver, a small mass was discovered attached to the trachea near its upper end. Further investigation revealed that the mass consisted of 2 contiguous tracheal diverticula. (The subject had died at the age of 76 of myocardial arrest and no further history was available.) Because of various unusual aspects of the specimen, it is described here with a brief review of the literature in the discussion.

DESCRIPTION OF SPECIMEN

Careful dissection of the mass, which originally was about the size and shape of a small hen's egg, revealed it to be formed by 2 contiguous, thick-walled, pedunculated diverticula, each communicating with the tracheal lumen through a tiny opening. They were adherent to the right posterolateral aspect of the trachea. After dissection, it was seen that each hung from the junction of the membranous and cartilaginous parts of the wall by a short pedicle—the upper one at the level of cartilaginous rings 5 and 6 and the lower one at the level of rings 7 and 8 (Figs. 1 and 2).

The upper diverticulum was the larger (measuring about 3 by 2.5 cm.) and had walls that were thick and fibrous looking. When it was squeezed there was a definite feeling of crepitation. Its pedicle was short and broad (about 7 mm.) and through it the cavity opened into the trachea by a tiny hole (less than 4 mm. in diameter).

In the wall at the lower pole of the upper diverticulum there was a firm inseparable lump. Histologically this lump contained many cystic spaces lined by a pseudostratified, ciliated, columnar epithelium of an embryonic type (Figs. 3 and 4). There was also a marked inflammatory reaction with many lymphocytes and considerable proliferation of fibrous tissue. There were no mucous glands, smooth muscle, or cartilage.

The smaller, lower diverticulum (1 by 1.5 cm.), although it was loosely adherent to the larger, did not communicate with it. It opened independently into the trachea by a tiny hole 1 cm. below the other through a pedicle which was somewhat longer and narrower than the other. Its interior was lined with smooth mucosa from which small evaginating pockets into the thick wall gave the wall a cystic, labyrinthine appearance.

The diverticula were not attached in any way to the neighboring thyroid gland which was somewhat enlarged. The right lower parathyroid gland was

Received for publication Jan. 11, 1960.

nicely preserved and easily seen in the specimen (Fig. 1). The esophagus was kinked to the left and partially compressed between the diverticula and the left lobe of the thyroid gland.

DISCUSSION

Tracheal diverticula (or tracheoceles) are usually thought of as being extremely rare and there are only scattered case reports in the literature (see

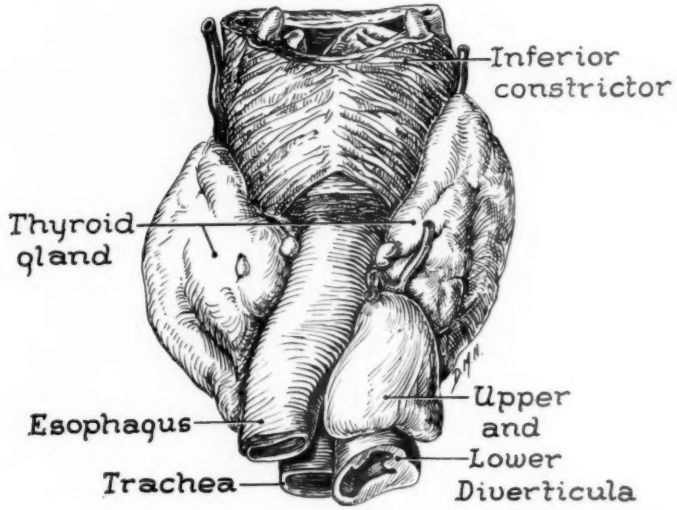


Fig. 1.—Specimen from behind. Lower diverticulum has been opened from below.

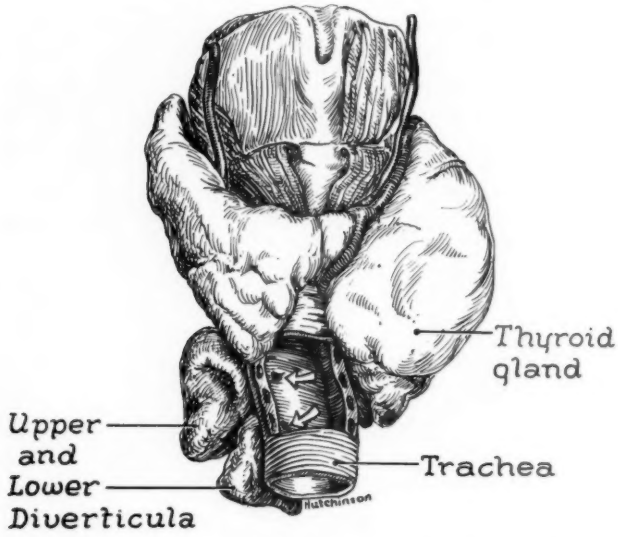


Fig. 2.—Specimen from in front. A window has been opened in wall of trachea and white arrows point to openings of upper and lower diverticula.

Goldman and Wilson[2]). However, MacKinnon[4] reported 8 cases very similar to ours in a series of 867 autopsies along with 2 other isolated cases. Chiari reported 6 cases at the lower end of the trachea, and Petit, in the nineteenth

century, had studied 41 cases (cited by Ziegelman[5]). It appears that the condition, although it well may be uncommon, is believed to be very rare only because it is often undetected.

Two views are held as to the etiology, the first being that the condition is congenital, and the second, that it is acquired. Rokitansky, who is generally conceded to have first described the condition in 1838, believed it to be retention cysts of the mucous glands secondary to infection in the upper part of the trachea and on the right side only (see Goldman and Wilson[2]). An explanation of this predilection for the right side has been given by several

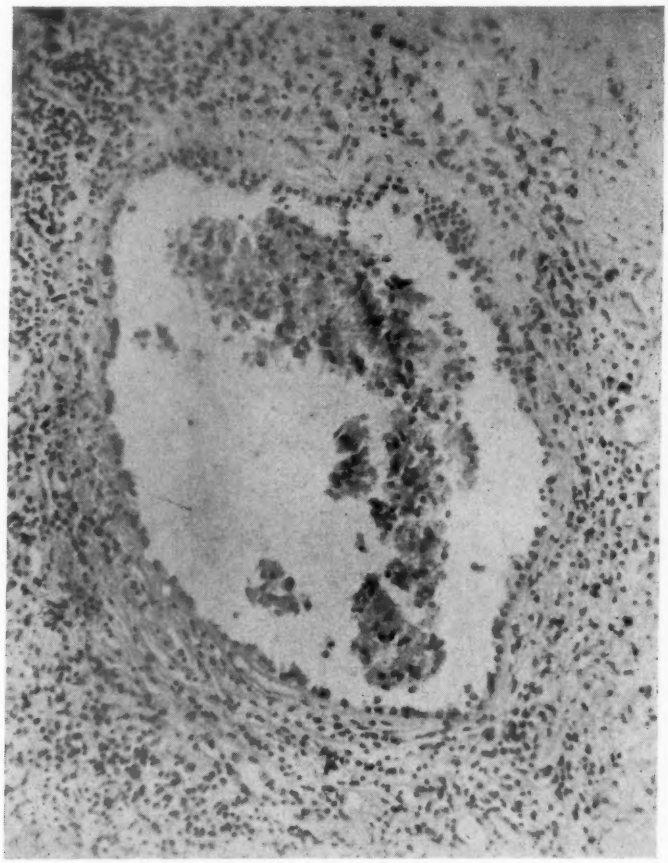


Fig. 3.—Low-power photomicrograph of one of cysts in the lump associated with the upper diverticulum. Much of the columnar epithelium is sloughed off. Note chronic inflammatory reaction in walls.

authors. Chiari (as quoted by Goldman and Wilson[2]) believed that the condition, being congenital, was a rudimentary bronchus on the right side at the site of a tripartition of the bronchial tree. MacKinnon,[4] however, explains the right-sidedness on the basis of the anatomic lack of support on the right side of the upper part of the trachea.

According to Ziegelman,[5] the acquired form is due to an outbulging at a weak spot in the posterior wall caused primarily by an infection of the mucous glands. Bremer,[1] on the other hand, supported his theory that the condition

is congenital by his finding that 5 per cent of young embryos have supernumerary tracheal bronchi which normally regress and disappear. Some may persist as rudiments and then become responsible for the formation of congenital and so-called spontaneous tracheoceles.

Lederer[3] believes that bronchogenic cysts associated with a tracheal diverticulum are congenital and result from a pinching off of a small bud or diverticulum of the foregut. When present, they are usually found on the posterior wall of the trachea near its bifurcation and are full of a clear, thick, jelly-like fluid. Their walls are lined with pseudostratified columnar epithelium and have a submucosa that contains mucous glands and cartilage.

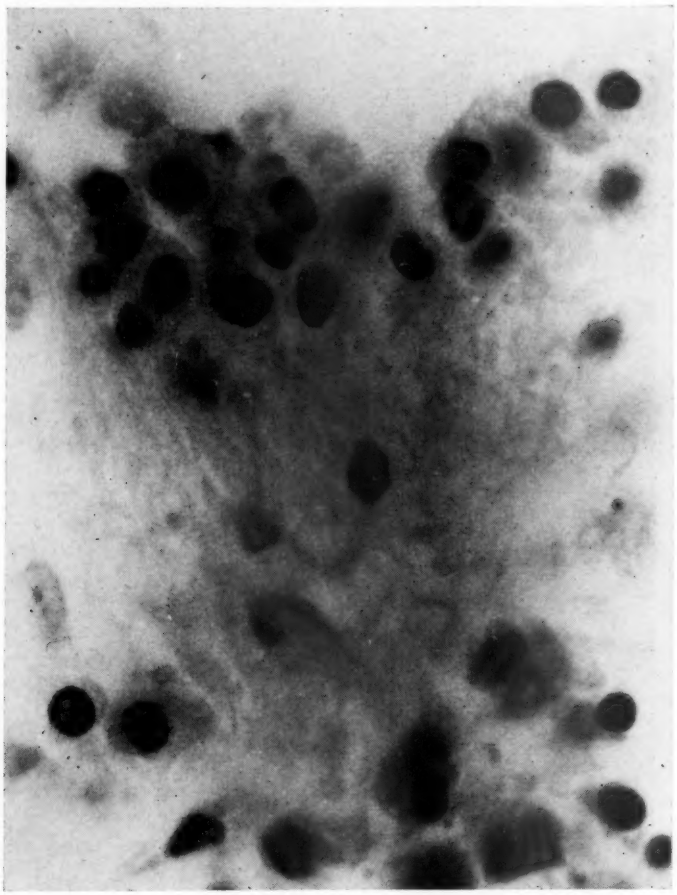


Fig. 4.—High-power photomicrograph of a small part of epithelium lining the cysts shown in Fig. 3.

The specimen reported in this paper might be claimed by the proponents of either theory to bolster their opinion. However, the weight of evidence makes us believe that the condition in this case is essentially a congenital one. It appears that 2 rudimentary tracheal bronchi have dilated or have been inflated and a cystic degeneration with secondary infection has occurred in the associated pulmonary tissue.

SUMMARY

A postmortem specimen of right-sided double diverticula from the back of the upper part of the trachea in a 76-year-old male is described. The literature, especially in regard to etiology, is reviewed and new doubt is cast on the general belief that this is an extremely rare condition. It is concluded that the cause, in this case at least, is essentially a persistence and then a dilatation or inflation of rudimentary supernumerary tracheal bronchi.

REFERENCES

1. Bremer, J. L.: Accessory Bronchi in Embryos; Their Occurrence and Probable Fate, Anat. Rec. **54**: 361, 1932.
2. Goldman, L., and Wilson, J. G.: Tracheal Diverticulosis, A. M. A. Arch. Otolaryng. **65**: 554, 1957.
3. Lederer, F. L.: Diseases of the Ear, Nose and Throat, ed. 6, Philadelphia, 1953, F. A. Davis Co.
4. MacKinnon, D.: Tracheal Diverticula, J. Path. Bact. **65**: 513, 1953.
5. Ziegelman, E. F.: Tracheal Diverticulum. Observations on a Cadaver and Results of Histological Study, Arch. Otolaryng. **21**: 414, 1935.

USE OF THE GRACILIS MUSCLE IN THE REPAIR OF A URETHROPERINEAL AND A VESICOPERINEAL FISTULA AFTER REMOVAL OF THE RECTUM

ALBERT MOWLEM, M.D., AND C. D. CREEVY, M.D., MINNEAPOLIS, MINN.

(From the Department of Surgery and the Division of Urology in the Medical School of the University of Minnesota)

FISTULAS of the bladder and urethra following abdominoperineal excision of the rectum are difficult to close because of lack of suitable tissue with which to make and buttress the repair. Most authors[5, 11] recommend immediate suprapubic cystostomy to divert the urine. Success is variable. If the injuries are overlooked at the time of the original operation, causing cystostomy to be performed late, failure is to be expected. Complicated operations and prolonged convalescence are likely to follow. Permanent urinary diversion by ureterosigmoidostomy or by implantation of the ureters into an isolated segment of ileum[1, 3, 7, 8] is especially unsatisfactory in patients with colostomies. These means of diversion are known to cause electrolyte imbalances[2, 6] and are unqualified nuisances from the patient's viewpoint.

The authors have recently treated a vesico- and a urethroperineal fistula successfully by what appears to be a new method. It occurred to one of us (C. D. C.) that the gracilis muscle would be ideal to re-enforce the usually inadequate closure of such fistulas, because it constitutes a readily available source of flexible tissue with a good blood supply.

CASE REPORT

A. S., a 46-year-old male, underwent a terminal colostomy and combined abdominoperineal resection for a large rectal cancer on Nov. 11, 1958. A portion of the prostate adjoining the cancer was excised, and the base of the bladder was lacerated, then closed by suture. A Foley catheter was left inlying. The regional lymph nodes were not involved. Ten days later urine began to drain from the posterior wound. Suprapubic cystostomy was performed on the sixteenth postoperative day and the patient was kept prone while suction was applied to the suprapubic tube. Urinary leakage continued.

Cystoscopy disclosed a fistula 1.5 cm. in diameter behind the vesical trigone. An excretory urogram was normal. In January, 1959, the posterior wound was reopened, curetted lightly, and kept packed with iodoform gauze until it had become lined with clean granulations.

On Jan. 30, 1959, the left gracilis muscle was divided near its insertion. It was mobilized up to the source of its blood and nerve supply. From here a subcutaneous tunnel was made in the anteromedial aspect of the thigh and in the contiguous perineum so as to enter the posterior wound. Insertion of a Van Buren sound into the urethra exposed a hitherto undetected defect 0.5 by 2 cm. in the floor of the bulbous urethra. Flaps were

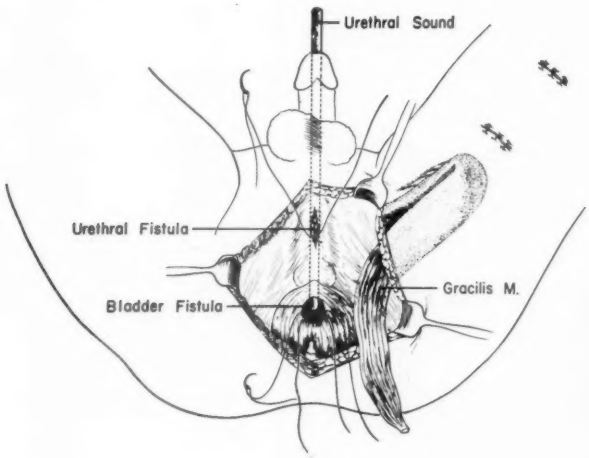
Received for publication Jan. 8, 1960.

fashioned from the scar tissue at its edges, and were united in the midline with interrupted No. 4-0 chromic catgut sutures (Fig. 1), because direct closure would have narrowed the urethra.

The edges of the vesical fistula were freshened and united with fine chromic catgut. Flaps of scar were then fashioned on each side of the closed fistula and united over it.

The gracilis muscle was drawn through the prepared tunnel and folded upon itself to fill the posterior space (Fig. 2). It was sutured loosely to the base of the bladder and to the

Fig. 1.



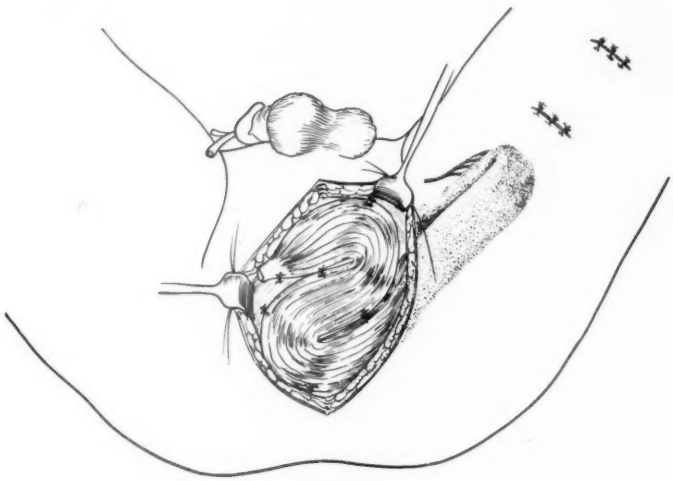


Fig. 2.

Fig. 1.—The gracilis muscle was mobilized from the left thigh and tunneled into the perineal space. Note the blood supply of the muscle in its proximal portion. The urethral and bladder fistulas may be seen. Repair was effected by using 2 layers made from scar tissue around them.

Fig. 2.—The gracilis muscle in posterior perineal space following repair of the fistulas. The skin edges were not approximated over the muscle during the operation.

bulbous urethra. The skin was left open. The patient was kept prone while suction was applied to the suprapubic tube, with an inlying urethral catheter used as an air vent. There was no leakage of urine thereafter. Both catheters were removed 4 weeks postoperatively, after which all urine was voided per urethram. Complete healing of the perineal skin required 2 months. Unfortunately, however, the urethral sphincters must have been injured during the

abdominoperineal operation, for the patient has experienced almost total urinary incontinence ever since. He can start and stop the stream upon command, but is unable to keep the sphincters contracted.

Three months after the fistulas had closed, cystoscopic examination, performed because of antibiotic-resistant pyuria, disclosed that there was no stricture at the site of urethral repair. There was a false passage into the posterior aspect of the prostate (Fig. 3). It is of interest that the anterior urethra looked normal and the site of repair was not detectable. Since it seemed likely that the intraprostatic pocket was maintaining the pyuria, the tissue between it and the prostatic urethra was excised with the resectoscope.

In a letter written 15 months after operation, the patient stated that, while his urine was clear (his physician found 8 to 10 white blood cells per high-power field), he had to wear a urinal because his control was insecure. He has been given instructions in sphincter exercises, but unfortunately he is not very faithful in carrying them out.

Fig. 3.—Urethrogram following repair of fistulas. Note irregularity of prostatic urethra because of false passage. The site of the repair of the urethra can scarcely be detected.

DISCUSSION

Four factors militate against the healing of urinary fistulas following removal of the rectum, (1) scarring of the affected structures, (2) the presence of an adjoining space which must fill by fibrosis, (3) the lack of local tissues which are flexible, mobile, and well supplied with blood, and (4) infection. The last need cause no concern if the wound is packed open until lined with healthy granulations.

Although the gracilis muscle has hitherto been used chiefly to make artificial sphincters in patients with neurogenic vesical or rectal dysfunction,[4, 9, 10, 12] the present case appears to represent its first application to the correction of traumatic perineal urinary fistulas of the bladder and urethra after removal of the rectum. The proximity of the gracilis to the perineum allows easy transplantation into the perineal (posterior) space. The fact that its blood supply and innervation enter its proximal one-fourth[9] permits mobilization and preservation of the distal three-fourths. The muscle is quite long, and constitutes a large mass of healthy, viable, soft tissue which may be adapted either as a sphincter or, as in the present case, as a buttress for the repair of urinary fistulas. Healing in our case was prompt and uneventful despite the approximation of flaps of scar over the fistulas. This demonstrates that as long as the right

kind of tissue is available, healing will usually occur. The situation is comparable to the use of a flap made of the rhomboid muscles and their blood supply to close successfully the open ends of chronically infected bronchi in patients with persistent bronchopleural fistulas following pulmonary resections.

The case is presented in order to re-emphasize the well-known surgical principle that healthy and vascular tissue is useful as a reinforcement in the repair of urinary fistulas. Also, in this case, the need for implantation of the ureters into the intestine with its inherent complications was obviated with gratifying results.

REFERENCES

1. Bricker, E. M.: Bladder Substitution After Pelvic Evisceration, S. Clin. North America **30**: 1511, 1950.
2. Creevy, C. D., and Reiser, M. P.: Observations Upon the Absorption of Urinary Constituents After Ureterosigmoidostomy, Surg. Gynec. & Obst. **95**: 589, 1952.
3. Coffey, R. C.: Physiologic Implantation of the Severed Ureter or Common Bile Duct Into the Intestine, J. A. M. A. **52**: 397, 1911.
4. Deming, C. L.: Transplantation of the Gracilis Muscle for Incontinence of Urine, J. A. M. A. **86**: 822, 1926.
5. Dodson, A. I.: Urological Surgery, St. Louis, 1956, The C. V. Mosby Company, chaps. 26 and 34.
6. Eiseman, B., and Bricker, E. M.: Electrolyte Absorption Following Bilateral Ureteroenterostomy Into an Isolated Intestinal Segment, Ann. Surg. **136**: 761, 1952.
7. Hinman, F.: A Simple Seven Suture Method of Bilateral Uretero-intestinal Implantation, Surg. Gynec. & Obst. **61**: 802, 1935.
8. Jewett, H. J.: Uretero-intestinal Anastomosis in Two Stages for Cancer of the Bladder, J. Urol. **52**: 536, 1944.
9. Pickrell et al.: Gracilis Muscle Transplant for Correction of Urinary Incontinence in a Male, Ann. Surg. **143**: 761, 1956.
10. Pickrell, K., Georgiade, N., Maguire, C., and Crawford, H.: Gracilis Muscle Transplant for Rectal Incontinence, SURGERY **40**: 349, 1956.
11. Praether, G. C.: Injuries of the Bladder, J. A. M. A. **154**: 205, 1954.
12. Smith, C. K., and Engel, L. P.: Neurogenic Vesical Dysfunction in Children, J. Urol. **28**: 576, 1932.

SIGMOIDOSCOPY AS PART OF ROUTINE CANCER CLINIC EXAMINATIONS WITH CORRELATED FECAL CHEMISTRY AND COLON CYTOLOGIC STUDIES

ALAN B. CAMERON, M.D., AND RAYMOND J. THABET, M.D., COLUMBUS, OHIO

(From the Departments of Surgery and Pathology, Ohio State University Hospital)

EACH patient examined in the general detection section of the Columbus Cancer Clinic during the past 2 years was scheduled for routine sigmoidoscopic examination. This study reports on the first 2,000 examinations. In addition to the usual statistics, data on the handling of routine outpatient sigmoidoscopies are discussed. Concomitant fecal chemistry and colon cytology are correlated with the sigmoidoscopic findings in approximately half of these patients.

SCHEDULING PROCEDURE

Each patient admitted to the general detection clinic was asked to return for a routine rectal examination in one of the 2 weekly sigmoidoscopic clinics. Lack of facilities and personnel prevented the giving of conventional enemas in the clinic. For this reason, packaged enema units were given to the patient. On the morning of examination he took the enema at home according to the instructions shown in Table I. Patients who lived far from Columbus were given the disposable enema on arrival for sigmoidoscopic examination. All instructions were given by the nurse scheduling the examination. To save time no further explanation of the test was given unless the patient requested it.

TABLE I. INSTRUCTIONS GIVEN PATIENT FOR PREPARATION FOR SIGMOIDOSCOPY

1. Eat a light breakfast
2. Take fluids only for lunch
3. Take enema 1 hour prior to appointment
4. Report to Cancer Clinic for examination at 1:00 P.M. on _____ (date)

Later in the study funds became available for conducting chemical and cytologic studies. Thus each of the last 850 patients was given a waxed container with a tight lid and was asked to bring a portion of his morning stool for examination.

SIGMOIDOSCOPY CLINIC PROCEDURE

Up to 25 examinations were scheduled for each afternoon sigmoidoscopy clinic. Fifteen hundred and seventy-nine of the examinations were performed by one author (A. B. C.). Stool specimens were collected on arrival of the patient. Prior to examination the sigmoidoscopist abstracted a short history from the patient's chart to a small record card. The sigmoidoscopist then

Received for publication Jan. 28, 1960.

explained the procedure to the patient as the latter was positioned by the nurse-assistant. Sigmoidoscopic results were put on the record card after the examination. Laboratory data and tissue and clinical diagnoses were added as they became available. These cards then became the records for this study.

Sigmoidoscopies were performed with the patient in knee-chest or left Sims' position. The former position was used generally. Sims' position was used for elderly patients who gave a history of severe heart trouble, pulmonary difficulty, or arthritis. Examinations averaged 7 minutes.

During the last 1,226 examinations, 2 cotton swabs of colonic mucosa were taken, 1 from the low rectum and 1 from the high sigmoid. In addition, swabs were taken from the surface of any mucosal lesion seen. Each swab was used to prepare a slide smear, which was immediately fixed in 95 per cent alcohol. These slides were stained and examined for malignant or unusual cells. The cytology report was then recorded on the patient's sigmoidoscopic card.

Biopsy, with a cold punch forceps was made of most lesions seen. Polyps were totally removed for biopsy. When polyps were too large for this procedure, the patient was advised to arrange for removal with his private physician. In all cases where polyps were found, the patient was advised to undergo 3 subsequent sigmoidoscopies at 3 month intervals, as well as an air contrast barium enema examination. He was then advised to have a sigmoidoscopic examination every 1 to 2 years.

Patients with malignant tumors were advised to arrange immediately with their family physician for treatment. In addition, a letter reporting the sigmoidoscopic and biopsy findings was sent to the physician.

Following examination the sigmoidoscopist discussed his findings and recommendations at appropriate length with the patient. If a biopsy had been performed, the patient was asked to remain for 1 hour as a check for rectal hemorrhage. Silver nitrate application was occasionally used to stop mucosal bleeding at time of biopsy.

RESULTS

Registrations at the Columbus Cancer Clinic are predominately by women. This is reflected in the 5 to 1 female to male ratio of patients in this study (Table II). Of patients scheduled, over 80 per cent appeared for examination.

TABLE II. SEX RATIO BY AGE

AGE RANGE	MALE	FEMALE	TOTAL
15-19	5	6	11
20-29	28	151	179
30-39	59	400	459
40-49	66	439	505
50-59	53	356	409
60-69	80	242	322
70-91	33	82	115
Total	324	1,676	2,000

In 85 patients colon cleansing was inadequate for sigmoidoscopy. In another 29 cases, sigmoidoscopy was unsatisfactory because of lack of patient

cooperation. The total number of unsatisfactory examinations represent 5.7 per cent of the number performed.

Of the 1,886 patients successfully examined, sigmoidoscopy was carried out to 23 to 25 cm. in 81 per cent of the cases. Ten per cent of patients were examined to less than 20 cm.

Eighty-six per cent of the patients had no symptoms referable to the colon. Table III lists the incidence of symptoms of the symptomatic group.

TABLE III. SYMPTOMS MANIFESTED BY PATIENTS*

SYMPTOMS	TOTAL NO. OF PATIENTS	NO. WITH BIOPSY OF POLYPS	NO. WITH MALIGNANT POLYPS	NO. WITH CARCINOMA
No symptoms	1,626	78	9	9
Rectal bleeding	115	12	1	5
Abdominal or rectal pain	87	9	0	0
Constipation	62	5	1	2
Diarrhea	41	2	0	1
Weight loss	6	2	0	0
Upper gastrointestinal symptoms	16	2	0	0

*Some patients (54) had more than one symptom.

In this study sigmoidoscopic examination was performed primarily to detect premalignant or malignant lesions of the rectosigmoid. Thirteen per cent of the patients had minor rectal pathologic findings, including hemorrhoids, fissures, or inflammation. In these cases no advice was given, but the patient was referred to his family physician for treatment.

Table IV gives the incidence of polyps and masses seen grossly. Seventy-seven per cent of the polyps of which biopsy was made gave histologic evidence

TABLE IV. STATISTICAL SUMMARY OF RECTOSIGMOID LESIONS OF 1,886 PATIENTS WITH TOTAL SATISFACTORY SIGMOIDOSCOPIES

	CONDITION	NO. OF PATIENTS	% OF 1,886
Total patients with polyps seen at sigmoidoscopy	Single polyps	103	
	Multiple polyps	9	
	Multiple polyposis	1	
	Total	113	5.99
Histologic diagnosis of patients with biopsy of polyps	Adenomatous polyps	60	
	Carcinoma in situ	11	0.58
	Colonic mucosa	18	
	Lymphoid hyperplasia	4	
	Squamous papillae	3	
	Villous adenoma	1	
	Total	97	
Histologic diagnosis of patients with mass	Adenocarcinoma	9	0.48
	Adenomatous change, rectal mucosa (mass probably extramucosal)	1	
	Chronic inflammation	1	
	Normal colonic mucosa (extramucosal mass)	2	
	Total	13	0.69
Total incidence of colonic carcinoma and malignant polyps			1.16

of adenomatous change. Of these, 14.3 per cent showed areas of carcinoma in situ. Considering these 11 polyps together with the 9 cases of carcinoma seen, there was a 1.2 per cent incidence of malignancy in this study.

Table V shows the location in centimeters of the benign and malignant polyps and carcinoma found.

TABLE V. LOCATION OF RECTOSIGMOID LESIONS

DISTANCE FROM ANUS (CM.)	CARCINOMA	MALIGNANT POLYPS	ADENOMATOUS POLYPS	POLYPS WITHOUT BIOPSY	NON-ADENOMATOUS POLYPS
1	-	-	-	-	3
2	1	1	-	-	2
3	-	1	-	-	1
4	-	-	2	1	-
5	1	1	1	-	-
6	-	-	5	-	1
7	1	-	-	1	-
8	2	-	3	-	1
9	1	-	1	-	-
10	1	-	8	1	2
11	-	-	1	-	-
12	-	-	5	1	-
13	1	1	7	3	1
14	-	1	5	2	2
15	1	3	11	1	4
16	-	-	7	1	2
17	-	-	5	-	-
18	-	1	7	-	1
19	-	-	-	-	-
20	-	2	7	1	3
21	-	-	-	-	-
22	-	-	2	-	1
23	-	-	1	2	-
24	-	-	1	1	1
25	-	-	2	1	-

Table III correlates symptoms with the presence of polyps or carcinomas. Of the 115 patients with gross rectal bleeding, 5 had carcinoma. Of the group with no colon symptoms 1 patient had a carcinoma and 78 had polypoid lesions, of which 8 were malignant.

TABLE VI. RESULTS OF STOOL BLOOD DETERMINATIONS OF 806* SPECIMENS

RESULTS	POSITIVE	OTHER
Normal sigmoid findings	54	505
Carcinoma	4	2
Malignant polyp	1	7
Benign polyp	4	49
Minor rectal findings	25	155
Total	88	718

*Specimens requested—850.

Eight hundred and six patients brought morning stool specimens which were semiquantitatively analyzed for blood by a benzidine hydrochloride method. The results are shown in Table VI. Eighty-eight patients gave an unequivocally positive reaction. Two of 6 patients with carcinomas showed no fecal blood. Seven of 8 patients with malignant polyps also were negative for

blood. Four of 53 patients with adenomatous polyps showed fecal blood. Seventy-nine patients, with no known colon pathology or minor rectal abnormalities, showed blood. Twenty-five per cent of these had hemorrhoids.

Mucosa smears were examined from 1,226 patients. Table VII shows the correlation between pathology found and the cytology report. In 12 instances malignant cells were seen. In 3 cases no further evidence of malignancy was found. In 5 a carcinoma in situ of a polyp or frank carcinoma of the colon was found. In the remaining patients 4 polyps were seen, but no biopsy was taken. One of these 4 was a patient with multiple polyposis. No carcinomas were missed by cytology. Five cases of malignant polyps, however, yielded no malignant cells. Seven of 35 cases of adenomatous polyps showed atypical cells suggestive of an adenomatous lesion.

TABLE VII. RESULTS OF CYTOLOGIC EXAMINATION FOR MALIGNANT CELLS, 1,226 PATIENTS WITH SPECIMENS

RESULTS	POSITIVE	OTHER
Normal sigmoid	3	0
Carcinoma	3	0
Malignant polyps	2	5
Papillary adenoma	1	0
Multiple polyposis	1	0
Polyps—no biopsy	2	28

DISCUSSION

More than 80 per cent of patients scheduled for sigmoidoscopy returned. In the surgical outpatient department of the Ohio State University Medical Center, less than 50 per cent of scheduled patients actually appear for such examination. The difference in motivation of these 2 groups presumably accounts for much of this discrepancy. Similarly, surgical outpatients failed to show the high compliance of Cancer Clinic patients with the request for fecal specimens.

Sigmoidoscopies were most easily performed on patients in the knee-chest position with the use of a proctologic table. The kneeling platform was positioned so that the patient's lower abdomen did not touch the examining table. This small detail seemed helpful in the performance of sigmoidoscopy, presumably by allowing freer movement of the pelvic viscera during examination. Full distance was reached in over 80 per cent of examinations. This figure could have been improved with greater patient cooperation. Since physician time is quite limited in this clinic, the value and procedure of sigmoidoscopy were not discussed with the patient until the time of examination. In addition, none of the patients knew the examiner. For these reasons patients were frequently quite nervous concerning the procedure, and cooperation was less than complete in many cases. However, patient compliance with the written instructions concerning presigmoidoscopic bowel preparation was excellent.

The fact that adequate colon cleansing was achieved in a high per cent of patients using self-administered disposable enemas was gratifying, especially in view of the nursing time saved. Self-administered enemas have greatly simplified both in- and outpatient sigmoidoscopy preparation and should encourage more routine office sigmoidoscopy.

The high percentage of benign and malignant polyps found in this study is not unusual.[1, 2] Of the 9 patients with carcinomas, only 1 was asymptomatic, but the symptoms of 6 of those remaining had not prompted them to seek medical care. Nine of the 11 patients with malignant polyps were also asymptomatic. These facts suggest that routine sigmoidoscopy is essential for early detection of premalignant and malignant lesions of the rectosigmoid.

Biopsy was taken of all polyps by total excision. In some cases (16) biopsy was not performed. In 3 cases, the polyp was too large to be excised as an outpatient procedure. In 7 cases, polyps were multiple and hospital admission was advised. The remaining 6 patients declined biopsy.

Stool tests for melena were begun in the hope that they might be useful in selecting patients for sigmoidoscopic examination.

Studies of the gum guaiac and benzidine hydrochloride reactions for fecal blood, by a semiquantitative technique, have shown that the gum guaiac reaction is positive in many cases where no fecal blood is present.[3, 4] For this reason the benzidine reaction results were used in this study. The test was made semiquantitatively to adjust for the sensitivity of benzidine to small amounts of fecal blood. Only those samples giving a strong reaction to benzidine hydrochloride were considered positive. If sigmoidoscopies had been performed only on patients with stool specimens positive for blood, the number performed would have been cut 90 per cent, and one-third of the carcinomas, as well as most of the benign and malignant polyps, would not have been found. Presently, then, this test for fecal blood is not acceptable for selecting asymptomatic patients for sigmoidoscopy.

Cytologic examinations of mucosa smears were performed to determine the frequency with which exfoliated cells from malignant growths or polyps could be identified. This method of sampling and cytologic examination has little practical application, since direct biopsy of lesions in this area is nearly always feasible and reliable. Most benign and malignant polyps did not yield identifiable cells by this method. Malignant cells, however, were demonstrated from each patient with malignant conditions of the rectosigmoid. This suggested that cytologic examination of colonic material might be useful in the detection of malignant growths in the colon. Recent workers[5, 6] have shown that malignant cells may be recovered from colonic washings from patients with carcinoma of the colon. However, a sigmoidoscope was inserted prior to collection of the cytologic material in these studies.

We have developed a method of recovering exfoliated malignant cells from enema returns, without instrumentation.[7] The possibility of using this test for selecting asymptomatic patients for definitive colon diagnostic procedures is now being investigated.

The cases of false positive identification of malignant cells in this study will be followed closely. One of these cases occurred in a patient with multiple polyposis, in whom, if not already present, the development of carcinoma of the colon is certain if colectomy is not performed.

Two patients in this study hemorrhaged sufficiently after biopsy to be admitted to the hospital. One of these had a 2 cm. polyp with a long stalk that

was removed completely. Removal of polyps of such size is no longer carried out in the Cancer Clinic because of lack of adequate cautery equipment. In the second patient, a 4 mm. sessile polyp was removed. The histologic diagnosis was adenocarcinoma in situ of the colonic wall. No adenomatous change was seen. No reason was found for the profuse hemorrhage from this small biopsy. Fortunately conservative measures controlled the hemorrhage in both these cases. No other complications of sigmoidoscopy or biopsy were encountered.

SUMMARY

A program of routine sigmoidoscopy for Cancer Clinic patients is described. Patient cooperation was excellent, especially in comparison with that of an outpatient clinic group. The value of such a program is shown by the fact that 1 patient of every 100 satisfactorily examined had a frank carcinoma of the rectosigmoid or a carcinoma in situ of a colonic polyp discovered on sigmoidoscopy. In addition, 70 cases of benign adenomatous polyps were found. Chemical determinations for fecal blood in 806 patients correlated rather poorly with the presence of carcinoma or polyps, either benign or malignant. Cytologic studies of colonic smears for malignant cells showed good correlation with the presence of carcinoma, but poor correlation with benign or malignant polyps. The value of routine sigmoidoscopy for early detection of premalignant and malignant lesions of the colon is emphasized.

REFERENCES

1. Portes, C., and Majarakis, J. D.: Proctosigmoidoscopy—Incidence of Polyps in 50,000 Examinations, J. A. M. A. **163**: 411-413, 1957.
2. Wilson, G. S., Dale, E. H., and Brines, O. A.: Symposium on Early Diagnosis of Tumors of Rectum and Colon; Evaluation of Polyps Detected in 20,847 Routine Sigmoidoscopic Examinations, Am. J. Surg. **90**: 834-840, 1955.
3. Cytologic Screening for Cervical Cancer, Comparative Findings in a Six Year Survey of a Well Population, Cancer **11**: 122-126, 1958.
4. Morgan, T. E., and Roantree, R. J.: Evaluation of Tests for Occult Blood in Feces, J. A. M. A. **164**: 1664-1667, 1957.
5. Bader, G. M., and Papanicolaou, G. N.: The Application of Cytology in the Diagnosis of Cancer of the Rectum, Sigmoid and Descending Colon, Cancer **5**: 307, 1952.
6. Raskin, H. F., Palmer, W. L., and Kirsner, J. B.: Exfoliative Cytology in the Diagnosis of Cancer of the Colon, Dis. Colon & Rectum **2**: 46-57, 1959.
7. Cameron, A. B., and Thabet, R. J.: S. Forum **10**: 30-33, 1959.

PARALYTIC ILEUS ASSOCIATED WITH PROLONGED BISHYDROXYCOUMARIN (DICUMAROL) THERAPY

CARLOS PEREZ-MESA, M.D., ST. LOUIS, MO.

(From the Division of Surgical Pathology, Department of Surgery, Washington University School of Medicine, Barnes Hospital, and the Barnard Free Skin and Cancer Hospital)

PROLONGED bishydroxycoumarin therapy rarely has been complicated by abdominal manifestations simulating acute intestinal obstruction. The symptomatology in such instances has been produced by intra-abdominal bleeding and diffuse serosal or interstitial hemorrhage in the small intestine wall. Data from patients with this complication reported in the literature and from 2 additional cases are presented. In these 6 instances, laparotomy was performed and the diagnosis was proved conclusively. No other lesions were found. Published cases[2, 3, 5, 6] that may represent the same type of complication, but in which no laparotomy was performed, were excluded from our study.

CASE REPORTS

CASE 1.—C. D., a 56-year-old man, was first admitted to Barnes Hospital on July 21, 1958, with angina pectoris. He received maintenance doses of Dicumarol for 27 hospital days. The prothrombin time averaged 20 per cent of normal. Medication was discontinued and the man was discharged from the hospital on Aug. 18, 1958. One week later the anginal pain recurred and he was readmitted to the hospital (Aug. 25, 1958). Dicumarol was reinstituted and he remained in the hospital until Sept. 17, 1958. During that time the lowest prothrombin time registered was 12 per cent of normal. Upon his discharge from the hospital he was instructed to continue taking 60 mg. of Dicumarol daily. After 59 days of Dicumarol medication, following minor trauma, the patient developed a subcutaneous hematoma over the entire left upper extremity. His prothrombin time was found to be 58 seconds at this time and the drug was discontinued and vitamin K given. Twenty-four hours later the patient developed lower abdominal cramping pain with nausea. By the next day the abdominal pain was severe, constant, and generalized. The patient was readmitted to Barnes Hospital on Oct. 23, 1958. On that day one stool was blood streaked. His temperature was 37.7° C., pulse 120, and blood pressure 144/88. The abdomen was tender in the lower quadrants and no masses were palpable. The bowel sounds were hyperactive. Roentgenograms (Fig. 1) showed distention of the small intestine and air in the ascending colon. The blood count was normal, hemoglobin was 12.2 Gm. and 15 to 20 red blood cells were found in the urinary sediment. Prothrombin time was 35 per cent of normal (22 seconds). Five hundred cubic centimeters of whole blood and vitamin K were given. The diagnosis of paralytic ileus was made. At laparotomy 600 ml. of unclotted blood was present in the peritoneal cavity. A hemorrhagic area in the jejunum was found, measuring about 30 cm. in length and involving the entire circumference of the bowel. Diffuse petechiae were seen over the intestinal serosa. The mesenteric vessels were normal. The hemorrhagic area was resected. The specimen (Fig. 2) showed a sharply circumscribed interstitial hemorrhage, thickening of the intestinal wall, but no noticeable reduction of the lumen. The neighboring intestine was edematous. On histologic examination the lesion consisted of extravasated blood in the submucosa infiltrating the inner muscular layers. The serosa, muscularis mucosa, and mucosa were intact, free of

Received for publication Feb. 8, 1960.

A. *B.* *C.*

Fig. 1.—Case 1. Extensive distention of the proximal small intestine with air fluid levels. Small amount of air is present in the large intestine with some fecal material in the ascending colon. The appearance is that of a massive regional paralytic ileus involving the proximal small bowel. Some intra-abdominal fluid is present.

inflammation and necrosis, and histologically viable. Edema and extreme capillary congestion without evidence of vascular rupture were present (Fig. 3).

The patient experienced no postoperative complications and was discharged from the hospital on the twenty-first postoperative day.

CASE 2.—S. G., a 67-year-old man with a 15-year history of angina pectoris, had been receiving Dicumarol daily for 14 weeks. Weekly prothrombin times averaged 11 per cent of normal. Two days before admission to the hospital the patient had severe chest pain radiating to the left shoulder which lasted for several hours. The next day there was

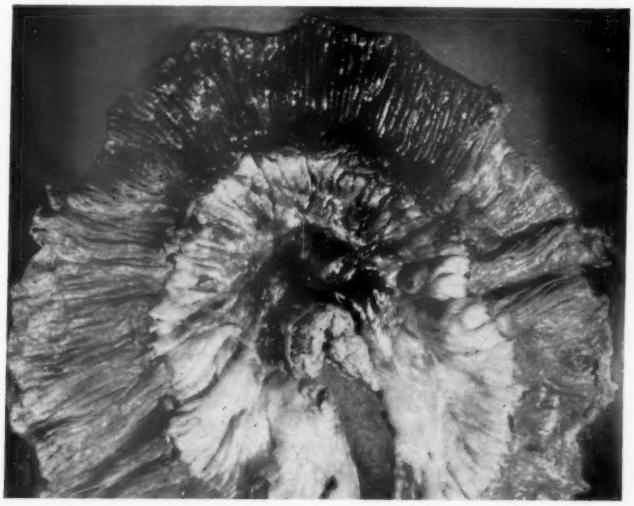


Fig. 2.—Case 1. Resected specimen showing the hemorrhagic area in the intestinal wall and in the mesenteric fat. Notice the preservation of the mucosa.

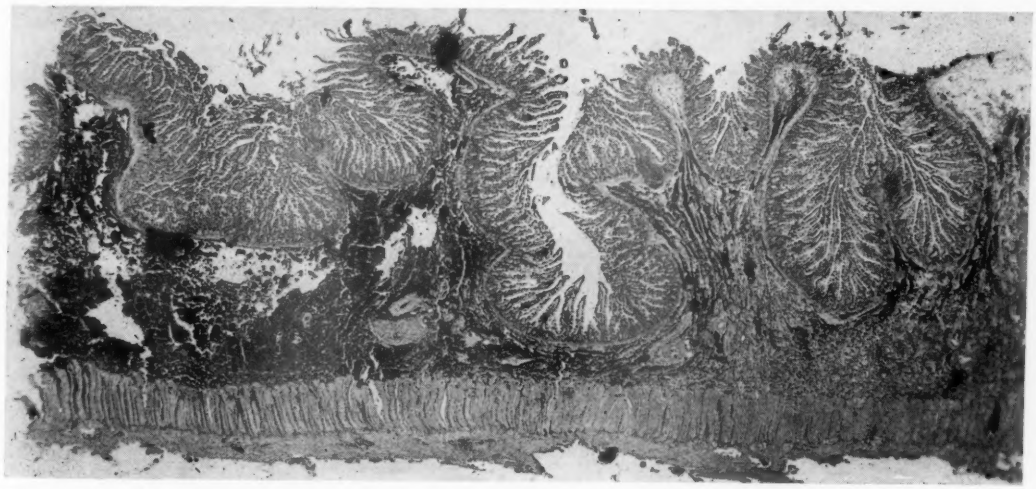


Fig. 3.—Case 1. Section through the hemorrhage showing the interstitial distribution with preservation of the mucosa. (×20; reduced ¼.)

abdominal pain in the lower quadrants, moderately severe and partially relieved by a bowel movement. Twelve hours later, the pain increased in severity and became generalized without nausea, vomiting, or diarrhea. The man was admitted to Barnes Hospital on March 10, 1957, at which time his temperature was normal, blood pressure 130/32, and pulse 92. The abdomen was distended and diffusely tender with rebound tenderness in the lower quadrants. No masses were palpated. Prothrombin time was 13 per cent of normal and urinalysis demonstrated rare red blood cells. The intestinal sounds decreased in activity over 10 hours of

TABLE I. DATA FROM LITERATURE AND TWO ADDITIONAL CASES

	AGE	SEX	INDICATIONS FOR THERAPY	DURATION OF TREATMENT BEFORE ONSET OF COMPLICATIONS	SYMPTOMS	BLEEDING MANIFESTATIONS
Mendelsohn, S., and Iglauer, M. (Case 1)	64	M	Pulmonary embolism	3 mo.	Abdominal pain, distention, no peristalsis heard, vomiting	Hematuria
Parrish, R. A.	72	F	Thrombophlebitis	11 mo.	Abdominal pain	None
Berman, H., and Mainella, F. S. (Case 3)	69	F	Myocardial infarction	33 days	Abdominal pain, tenderness, vomiting, fever	Not stated
Pearson, S. C., and McKenzie, R. C.	69	M	Thrombophlebitis	Not stated (last month of treatment was without laboratory control)	Abdominal pain, distention, no peristalsis heard	Bloody vomiting
Author's Case 1	56	M	Myocardial infarction	2 mo.	Abdominal pain, distention, nausea	Skin petechiae, rectal bleeding
Author's Case 2	67	M	Myocardial infarction	3 mo.	Abdominal pain, distention, nausea, decreasing intestinal sounds	None

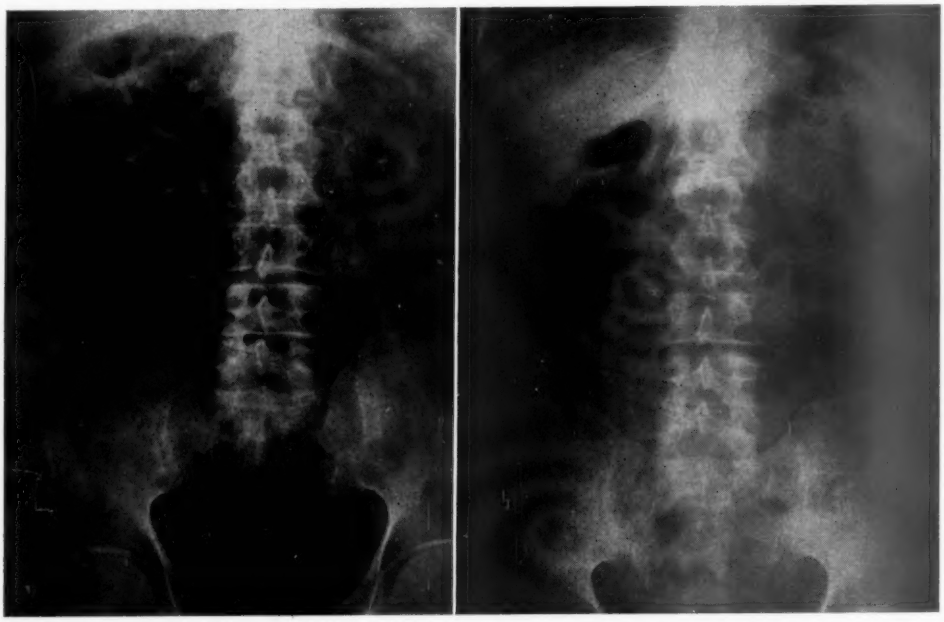


Fig. 4.—Case 2. Roentgenograms showing distention of the large and small bowel involving predominantly the lower jejunum and proximal ileum. The appearance is that of a regional paralytic ileus located predominantly in the right upper abdomen.

OF PARALYTIC ILEUS AFTER PROLONGED DICUMAROL THERAPY

LENGTH OF PROTHROMBIN TIME (SECONDS)	X-RAY CHANGES	DIAGNOSIS AT ADMISSION	OPERATIVE FINDINGS	TREATMENT AND COURSE
Not stated	Dilatation of small intestine	Acute abdominal crisis	Hemoperitoneum, diffuse serosal petechiae of the small intestine	No resection, vitamin K and fresh blood, uneventful recovery
72	Dilatation of small intestine	Intestinal obstruction	Hemoperitoneum, serosal petechiae in ileum and jejunum	No resection, vitamin K and fresh blood, uneventful recovery
30	Dilatation of proximal loops of small intestine	Acute abdominal crisis	Hemoperitoneum, interstitial hemorrhage in jejunum 25 cm. length, diffuse serosal petechiae	Resection of the hemorrhagic jejunal segment, uneventful recovery
Under 10% of normal	Dilatation of small intestine	Intestinal obstruction	Hemoperitoneum, interstitial hemorrhage in jejunum 20 cm. length	Resection of the hemorrhagic jejunal segment, uneventful recovery
58	Dilatation of small intestine	Intestinal obstruction	Hemoperitoneum, interstitial hemorrhage in jejunum 30 cm. length	Resection of the hemorrhagic jejunal segment, uneventful recovery
	Dilatation of small intestine	Intestinal obstruction	Hemoperitoneum, interstitial hemorrhages in jejunum 13 and 8 cm. in length	Resection of the jejunal hemorrhagic areas, uneventful recovery

observation. Roentgenograms (Fig. 4) showed dilated loops of small intestine with air fluid levels and evidence of free peritoneal fluid. The diagnosis of paralytic ileus was made. Five hundred cubic centimeters of whole blood and vitamin K were given. Immediately before the operation the prothrombin time was 50 per cent of normal. At laparotomy 250 ml. of unclotted blood were present in the peritoneal cavity. Two hemorrhagic segments of jejunum measuring 8 and 13 cm. in length, respectively, and separated by an intervening 10 cm. of normal intestine, were found. Petechiae were present throughout the mesenteric serosa. The mesenteric vessels were normal. The hemorrhagic areas of bowel were resected and a jejunojejunostomy was performed. The resected specimen consisted of 40 cm. of jejunum including the 2 hemorrhagic segments and intervening normal intestine. The gross and microscopic changes were similar to those described in the first case. The patient had an uneventful postoperative course.

Data collected from the literature and from our 2 cases are given in Table I.

DISCUSSION

The earliest complication occurred after 33 days of Dicumarol therapy. In all cases there was a rather sudden onset of cramping abdominal pain with nausea and vomiting. Abdominal distention was present in all cases. In one of our patients (C. D.) the intestinal sounds were hyperactive. Localized or diffuse abdominal tenderness was the rule. In some instances there was evidence of intraperitoneal fluid. Roentgenograms were suggestive of paralytic ileus and showed dilated loops of small bowel with air fluid levels. Although paralytic ileus was the initial clinical diagnosis in all cases, other diagnoses

suggested were mesenteric thrombosis, appendicitis, and pancreatitis. At laparotomy hemoperitoneum was invariably found. In 2 instances[3, 5] there were petechiae on the entire serosal surface of the small intestine without evidence of an isolated hemorrhagic intestinal segment. The mesenteric vessels were normal but the mesentery was edematous. No resection was carried out in these 2 patients and subsequent recovery was uneventful.

In the other 4 patients[2-4] hemorrhagic jejunal segments up to 30 cm. in length were found in addition to the hemoperitoneum and serosal petechiae. Grossly, these segments were considered nonviable and resection was carried out. The mesenteric vessels were normal in all cases. The resected specimens all showed submucosal interstitial hemorrhage situated between the inner muscular layer of the muscular wall and the muscularis mucosa. The lamina propria and the mucosa were intact. No evidence of necrosis and no inflammatory infiltrate were seen.

SUMMARY

The development of signs and symptoms simulating intestinal obstruction in patients receiving Dicumarol should suggest that hemorrhage into the peritoneal cavity may have occurred, possibly combined with segmental interstitial hemorrhage into the intestinal wall. No characteristic clinical findings were noted that would suggest the extent or the location of the bleeding or that would rule out the existence of other intra-abdominal conditions. In no case was the hemorrhage of massive proportions. Although no evidence of necrosis was found in the hemorrhagic areas in the intestine, it is not known what the fate of these segments would be with conservative or nonoperative management. With respect to morphology, no specific lesions attributable to Dicumarol were recognized.

REFERENCES

1. Benedetti-Valenti, S.: Sindrome addominale acuta, atipo di occlusione intestinale nella intossicazione Dicumarolica, Minerva chir. 9: 1179-1181, 1954.
2. Berman, H., and Mainella, F. S.: Toxic Results of Anticoagulant Therapy, New York J. Med. 52: 725-727, 1952.
3. Mendelsohn, S. M., and Iglauer, M.: Acute Abdominal Condition Due to Bis-Hydroxycoumarin (Dicumarol) Poisoning, A. M. A. Arch. Int. Med. 92: 760-762, 1953.
4. Paraf, A., and Paolaggi, J.: Syndrome abdominal aigu au cours d'une intoxication par le Dicoumarol, Semaine Hôp. Paris, 31: 1989-1990, 1955.
5. Parrish, R. A.: Intestinal Obstruction. A Complication of Anticoagulant Therapy, Am. J. Surg. 97: 787-788, 1959.
6. Pearson, S. C., and MacKenzie, R. J.: Intestinal Obstruction Due to Bishydroxycoumarin Poisoning, J. A. M. A. 167: 455-456, 1958.

LUMBAR AMPUTATION OR HEMICORPORECTOMY FOR ADVANCED MALIGNANCY OF THE LOWER HALF OF THE BODY

CHARLES S. KENNEDY, M.D., ELMER B. MILLER, M.D., DONALD C. MCLEAN, M.D., MARVIN S. PERLIS, M.D., RAYMOND M. DION, M.D., AND VICTOR S. HORVITZ, M.D., DETROIT, MICH.

(From the Division of Surgery and the Division of Medicine, The Grace Hospital)

IN 1948, one of us saw a female patient who, after thorough study had been made, was considered to have no apparent disease except in the pelvis. This woman had a previously x-ray treated carcinoma of the cervix uteri. At exploratory celiotomy it was obvious that both lateral pelvic walls were involved by carcinoma. The malignancy, however, was so limited that it was also apparent that removal of the lower half of the body through the midlumbar area would possibly have cured the patient. After the exploratory operation, the possibility of such an amputation was discussed with her internist and family physician. They immediately requested other surgical consultants and recalled the radiologist to the case.

The suggested amputation was deemed inhumane even if possible, which was doubted. The possibility of having the woman's life saved by such an operation was never mentioned to the patient, and she suffered for a year and a half with slowly progressing carcinoma until she died in uremia. Her treatment had consisted of further x-ray therapy and narcotics. She had had almost constant pain and constant foul vaginal discharge. She had once been a courageous, optimistic, kind, and cheerful person and had ended as a wailing nonentity, whose death was a relief to all concerned.

All of us have seen similar patients in the intervening years, but the circumstances did not move us to suggest to such patients the possibility of lumbar amputation or hemicorporectomy until the case to be presented occurred. The operation was discussed and no decision could be reached by us although the patient urged us to proceed. It was at this point that one of us talked with Dr. Alexander Brunschwig of New York who gave us the encouragement necessary to carry out the operation.

CASE REPORT

J. L., a 74-year-old white man, was first seen on June 18, 1959. He had had rectal bleeding after bowel movements for 1 year. On further questioning, the patient reported a 10 pound weight loss during the past month and difficulty moving his bowels and starting the urinary stream. A hemorrhoidectomy had been performed 1 year before because of rectal bleeding.

Physical examination showed the man to be generally healthy, except for a fungating mass just inside the anus and diffuse prostatic enlargement.

He was admitted to The Grace Hospital on June 21, 1959. Biopsy was performed on the rectal mass on June 23 and it was reported as adenocarcinoma, grade 2. After complete preparation, an abdominoperineal excision was carried out on July 3 (Fig. 1). At operation

Received for publication May 12, 1960.

there was no gross evidence of spread beyond the rectum, but microscopic section revealed adenocarcinoma, grade 2, of the rectum with extension through the wall without demonstrable lymph node metastases.

Initially, the postoperative course was uneventful except that the patient was unable to void voluntarily at any time. On July 22, a suprapubic prostatectomy was performed with a microscopic diagnosis of benign adenomatous hypertrophy of the prostate gland.

The patient progressed satisfactorily for 1 week, but on July 30 developed severe chest pain with râles and cough. X-ray changes of pulmonary embolism were observed. Anticoagulant therapy was started, and the prothrombin activity was maintained between 15 to

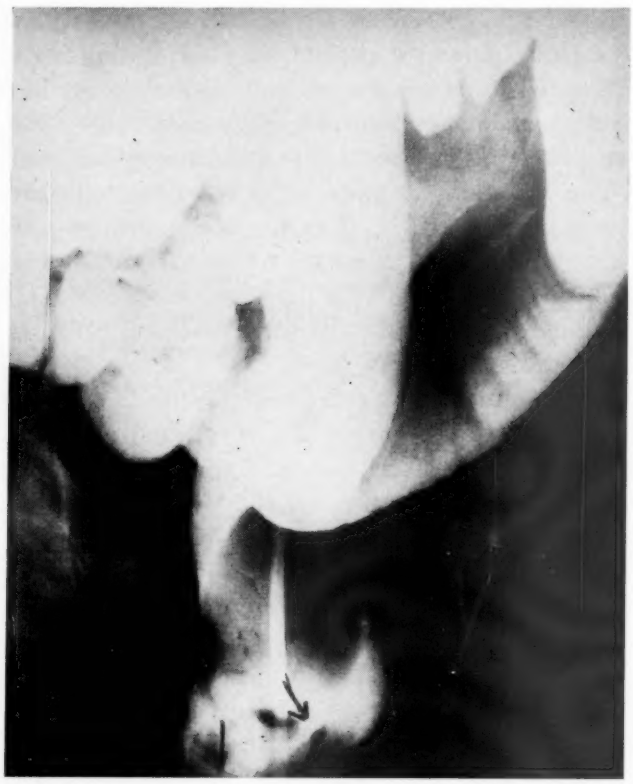


Fig. 1.—Barium enema preceding the abdominoperineal excision. The film demonstrates a large filling defect (arrows) due to carcinoma low in the rectum. The defect above is due to the balloon on the rectal catheter.

20 per cent of normal. In spite of this, further showers of emboli occurred on August 1 and August 8. On August 10, an inferior vena caval screen* was constructed and no further embolization occurred. The patient regained strength and was discharged in good condition on Sept. 10, 1959.

He was readmitted to the hospital on Nov. 24, 1959, with the complaint of high fever and increased frequency and burning of urination of 2 weeks' duration. Examination revealed much recent weight loss, a normally functioning colostomy, and a draining perineal wound. *Staphylococcus aureus* was cultured from the urine, and moderate right hydronephrosis demonstrated (Fig. 2, *A*). Antibiotic therapy was instituted, and the patient was discharged on December 6 after having normal temperatures for 1 week. The draining perineal wound was not treated at this time.

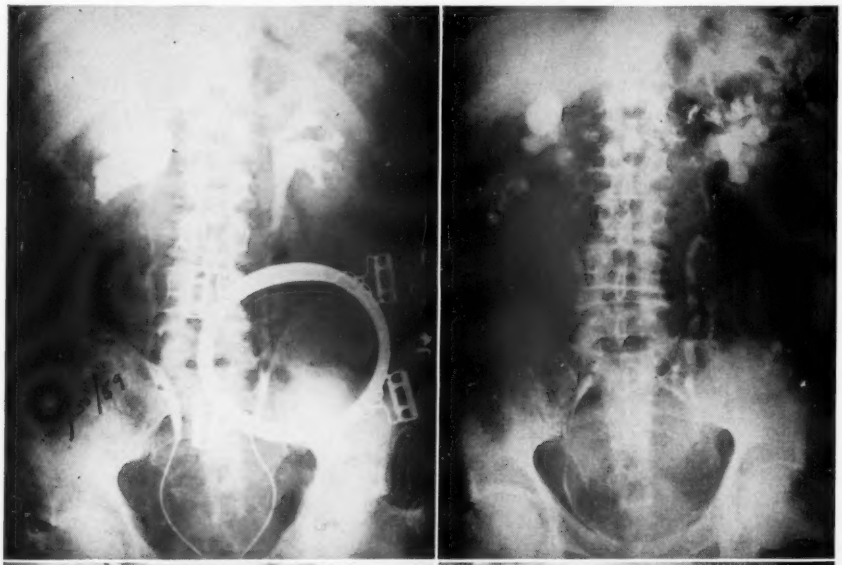
The final admission to the hospital was on Jan. 29, 1960. Drainage, from the perineal wound was profuse, with a permeating foul odor. The man's weight had declined 7 pounds since the previous hospital stay, his appetite was poor, and he was quite withdrawn because

*To be reported elsewhere.

of the odor of the drainage. His general condition appeared good, however, and there was no evidence of carcinoma except in the pelvis.

On February 3, a biopsy of the perineum was performed which showed an adenocarcinoma, grade 2, invading the skin and subcutaneous tissue. Cystoscopy showed a dis-

A. Fig. 2. *B.*



A. Fig. 3. *B.*

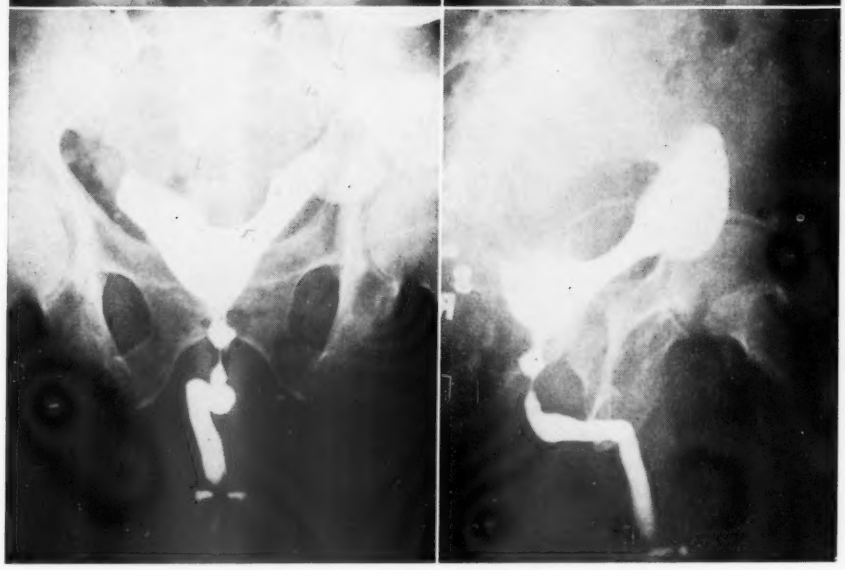


Fig. 2.—Pyelograms (*A*, Nov. 30, 1959, and *B*, Feb. 8, 1960) showing progressive evidence of bilateral urinary tract obstruction. Pyelograms had been normal preceding resection of prostate gland in July, 1959.

Fig. 3.—*A*, Anterior-posterior and *B*, lateral views of a cystogram taken 1 week before the hemicorporectomy. The films illustrate a gross distortion of the urinary bladder by the pelvic mass.

placed trigone with tumor invasion, and biopsies of the bladder were diagnosed as adenocarcinoma. Cystopyelograms at this time revealed a bilateral hydronephrosis (Figs. 2 and 3). Clinically there was a continuous drainage of urine through the perineal wound, and the patient reported that he had passed stools through it on several occasions. Fistulas were not demonstrable roentgenographically, however.

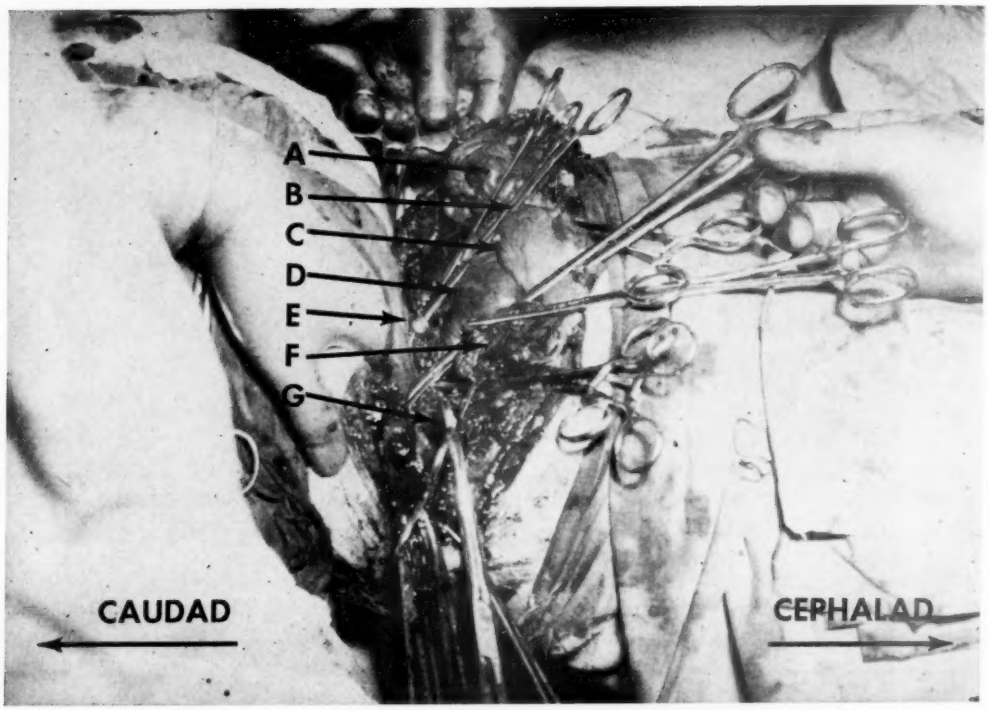


Fig. 4.—Stage of the operation with completion of the ileo-ascending colostomy cephalad to the isolated ileum for the ureteroileostomy. *A*, Ascending colon, *B*, ileocolostomy, *C*, ileum, *D*, isolated ileum, *E*, right ureter, *F*, proximal portion of sigmoid colon, *G*, left ureter.

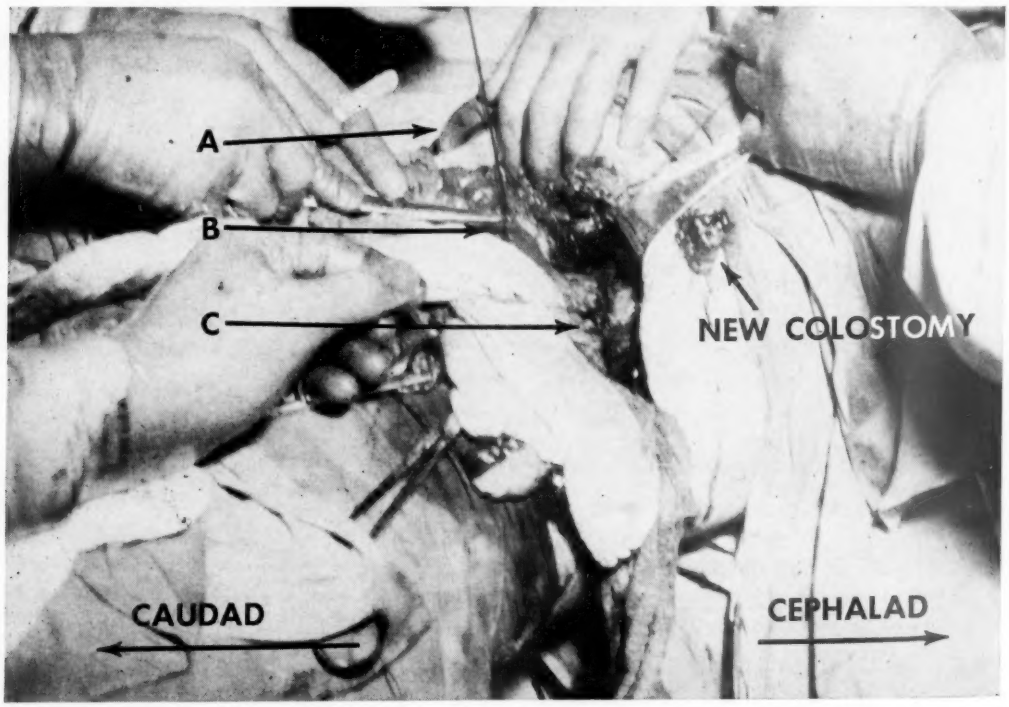


Fig. 5.—In this photograph the new colostomy has been established. *A*, Isolated segment ileostomy, *B*, completion of right ureteroileostomy, *C*, completed left ureteroileostomy.

At this point the patient was consulted, and the exact nature of his condition explained. The various forms of chemotherapy and x-ray treatment were mentioned as means of palliation. Because of the extensive invasion of the tumor in the pelvis, surgical palliation in the ordinary sense was deemed impossible. The patient stated that he could no longer tolerate the foul drainage from the pelvis and said he was willing to undergo any extensive procedure which would rid him of the malignancy. The feasibility of hemicorporectomy was then considered, and Dr. Brunschwig was consulted. It was generally agreed that the man should undergo exploratory operation, and if no gross metastases were found in the upper abdomen, a hemicorporectomy would be carried out. This was again explained to the patient, and he agreed that the operation should be attempted.

On Feb. 13, 1960, he was anesthetized with intravenous Pentothal sodium gas-oxygen-ether and a transverse incision was made in the lower abdomen. Exploration revealed no gross evidence of metastasis in the abdomen above the solid pelvic mass. The terminal ileum,

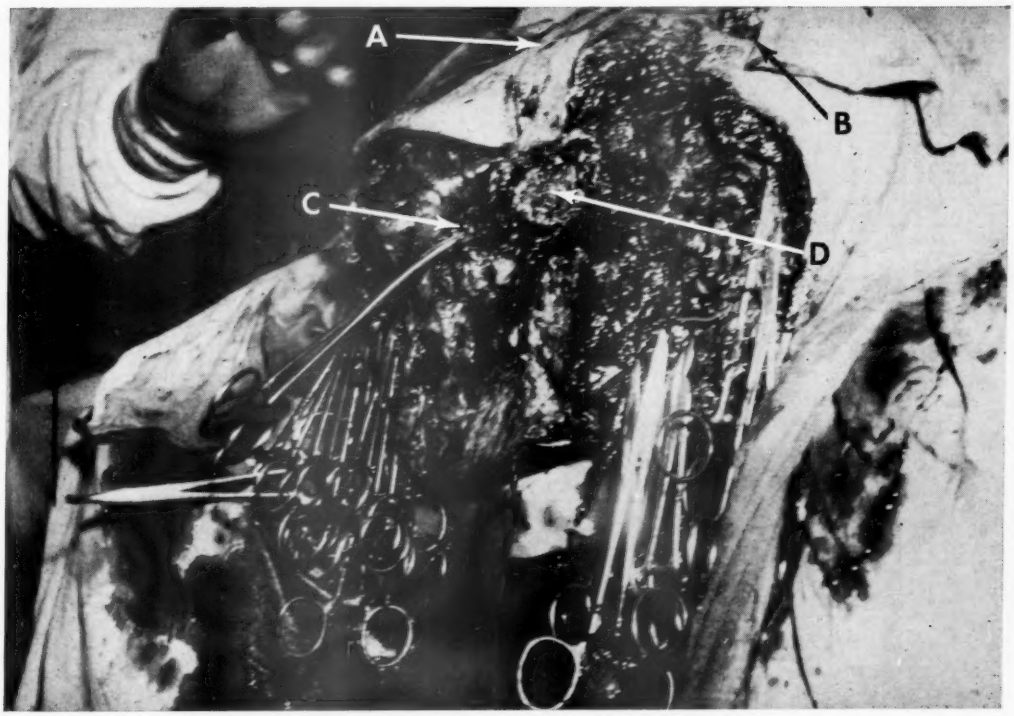


Fig. 6.—*A*, Ileostomy, *B*, colostomy, *C*, suture line of the anterior rectus muscle sheath to the lumbodorsal fascia, *D*, body of the third lumbar vertebra.

the appendix, and the tip of the cecum were involved in this mass. Therefore, the terminal ileum, cecum, and the sigmoid colostomy were resected. The dilated ureters were resected above the tumor, anastomosed into an isolated segment of terminal ileum, and a cutaneous ileostomy performed on the right side of the abdomen at a point just superior to the umbilical level. A terminal left colostomy was made at the same level. A side-to-side ileo-ascending colostomy was then performed to complete the continuity of the gastrointestinal tract (Figs. 4 and 5). The peritoneal cavity was closed by approximation of the anterior and posterior peritoneal layers. The vena cava was ligated and severed, as were the common iliac arteries. The lumbar muscles were divided, the lumbar vessels ligated, and the body disarticulated between the third and fourth lumbar vertebra. This was done by removing vertebral body No. 4, opening into the spinal canal, and ligating the dural sac and cauda equinae with a mass ligature of silk to prevent leakage of spinal fluid. Fig. 6 shows the completion of the amputation. The abdominal fascia was sutured to the lumbar fascia, and the skin of the abdomen and back were approximated with interrupted sutures.

The removed specimen consisted of everything below the level of the third lumbar vertebra. Gross pathologic examination showed a solid pelvic mass invading all major pelvic vessels and nerve bundles. There were ileoperineal, cystoperineal, and sigmoidoperineal

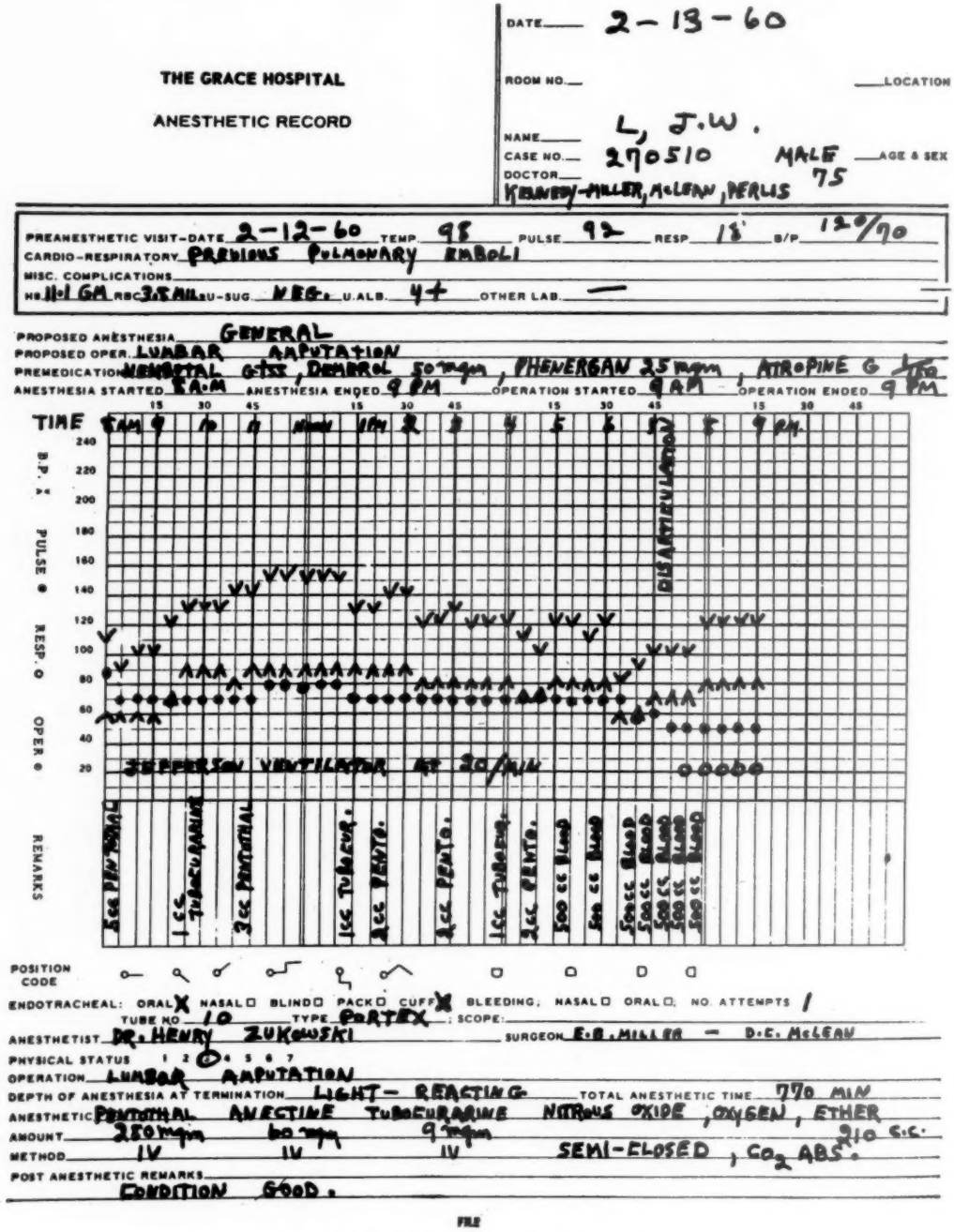

THE GRACE HOSPITAL
ANESTHETIC RECORD
DATE 2-13-60
ROOM NO. LOCATION
NAME L, J.W.
CASE NO. 270510 MALE AGE & SEX 75
DOCTOR KENNEDY-MILLER, McLEAN, MERLIS
PREANESTHETIC VISIT-DATE 2-12-60 TEMP 98 PULSE 92 RESP 18 B/P 120/70
CARDIO-RESPIRATORY PREVIOUS PULMONARY EMBOLI
MISC. COMPLICATIONS
HB 11.1 GM RBC 3.8 MILL U-SUG NEG. U.ALB 4+ OTHER LAB —
PROPOSED ANESTHESIA GENERAL
PROPOSED OPER LUMBAR AMPUTATION
PREMEDICATION NEMBUTAL GRS, DEMEROL 50 mgm, PHENERGAN 25 mgm, ATROPINE G 1/150
ANESTHESIA STARTED 8 A.M ANESTHESIA ENDED 9 PM OPERATION STARTED 9 AM OPERATION ENDED 9 PM
TIME
B.P.
PULSE
RESP.
OPER.
REMARKS
JEFFERSON VENTILATOR AT 20/MIN
ARTERIOGRAM
POSITION CODE
ENDOTRACHEAL: ORAL X NASAL BLIND PACK CUFF X BLEEDING: NASAL ORAL NO. ATTEMPTS 1
TUBE NO 10 TYPE PORTEX SCOPE:
ANESTHETIST DR. HENRY ZUKOWSKI SURGEON E.B. MILLER — D.E. McLEAN
PHYSICAL STATUS 1 2 3 4 5 6 7
OPERATION LUMBAR AMPUTATION
DEPTH OF ANESTHESIA AT TERMINATION LIGHT — REACTING TOTAL ANESTHETIC TIME 770 MIN
ANESTHETIC PENTOTHAL ANECTINE TUBOCURARINE NITROUS OXIDE, OXYGEN, ETHER
AMOUNT 250 mgm 60 mgm 9 mgm 210 c.c.
METHOD IV IV IV SEMI-CLOSED, CO_2 ABS.
POST ANESTHETIC REMARKS
CONDITION GOOD.
FILE

Fig. 7.—Condensed anesthetic record.

fistulas, almost complete replacement of bladder with tumor, and almost complete obstruction of the ureters with marked hydroureters.

The operative procedure lasted 13 hours and required 3,500 c.c. whole blood, 1,000 c.c. of 5 per cent glucose in water, and 100 mg. intravenous hydrocortisone. The blood pressure remained stable throughout the operation. Fig. 7 is the condensed anesthetic record and

Fig. 8 is a condensed chart of temperature, pulse, and respiration. Postoperatively the patient was maintained on intravenous fluids for 3 days and was placed on oral feedings when flatus passed freely through the colostomy. The urinary output was adequate through

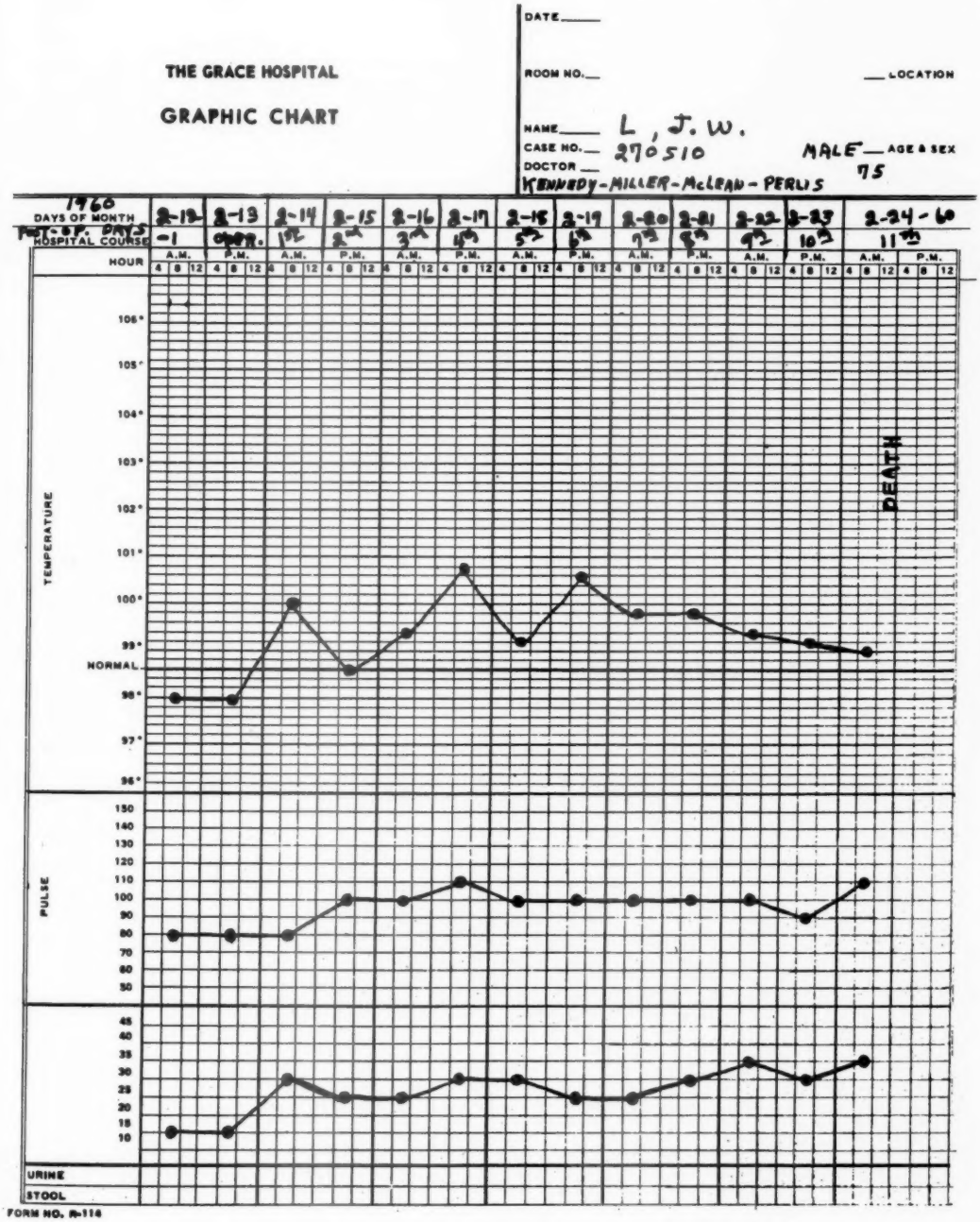


Fig. 8.—Condensed temperature, pulse, and respiration chart.

the cutaneous ileostomy. Intake and output are charted in Fig. 9. Blood urea nitrogen was 50 mg. per cent on Feb. 15, 1960, and decreased to 26 mg. per cent on February 23. Blood electrolytes remained normal, and on February 23 were: plasma chlorides 103 mEq., serum sodium 138 mEq., serum potassium 4.3 mEq., and CO_2 combining power 24.8 mM. or 55 vol. per cent. Postoperatively the red blood count was 3.6 million with 11.7 Gm. of

hemoglobin and decreased to 2.5 million with 8.3 Gm. of hemoglobin on February 21. Table I gives the results of postoperative laboratory tests.

Blood transfusion seemed indicated and on February 22, 250 c.c. of blood was given, with another 250 c.c. administered on February 23. Immediately following the second transfusion, the patient became dyspneic with wheezing, which seemed to subside in about 3 hours.

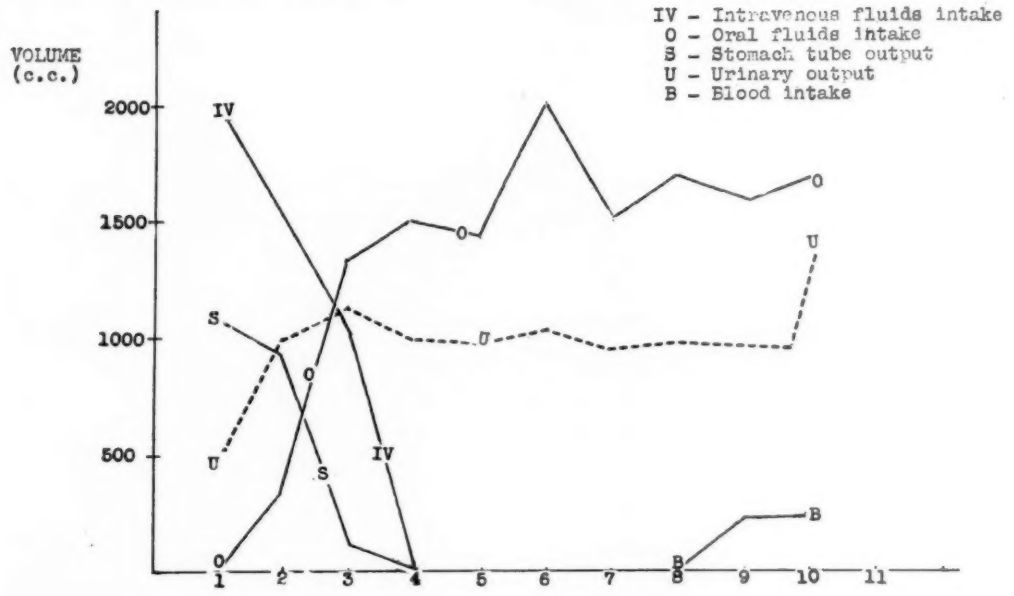


Fig. 9.—Intake-output chart.

TABLE I. LABORATORY CHART

LABORATORY TEST	PREOPERATIVE VALUE	POSTOPERATIVE DAY 1	2	3	4	5	6	7	8	9	10	11
Hb (Gm.)	11.1	13.8	13.7	11.7					8.0	8.3		
RBC (million)	3.8		4.1	3.6					2.5	3.0		
WBC	10,500			16,800						7,600		
BUN (mg. %)	51		50			30					26	
Blood Cl (mEq.)			82			103					110	
K (mEq.)			4.9			4.3					3.7	

At 5:30 A.M. on February 24, he developed a coughing spell, became cold, clammy, and dyspneic, and in spite of all emergency measures, died at 8:05 A.M.

Autopsy revealed pulmonary edema bilaterally with some stenosis and scarring of the right bronchus. There were no abnormal findings in the abdomen, brain, or spinal canal.

DISCUSSION

We believe that removal of the lower half of the body should be offered to the patient forthrightly in such a case as described when it is apparent that no

chance for survival exists with any procedure short of lumbar amputation and when such an amputation offers a hope of cure or of great relief of symptoms.

Our experience demonstrates the technical feasibility of the operation and points out only too clearly the importance of meticulous postoperative care. This patient was doing well postoperatively (Fig. 10), and it is the belief of all of us that he might have recovered if the possible error in judgment had not been committed which led to the second 250 c.c. blood transfusion on the tenth postoperative day.

Fig. 10.—The patient on the tenth postoperative day supporting his body free from the bed with his hands on an overhead trapeze. The catheter is in the ileostomy. His spirits and personal outlook for the future were excellent at this time.

CONCLUSION

Hemicorporectomy has been accomplished for otherwise hopeless malignancy. The patient survived to the eleventh postoperative day, and it was believed by all of us that he would have recovered had not his blood volume been unduly increased. All of us agree in retrospect that the operation was indicated and that we will proceed with it again when the circumstances arise.

The postmortem examination failed to demonstrate any residual carcinoma, and the cause of death was stated as pulmonary edema subsequent to failure of the left side of the heart. An incidental finding of interest was an embolus caught by the vena cava screen.

Postoperatively, the patient was comfortable, cheerful, and certain of his own rehabilitation. He actually sought to reassure us and was concerned about our feelings as well as his own. His condition was evaluated postoperatively by Dr. Joseph N. Schaeffer of the Rehabilitation Institute of Metropolitan Detroit, who believed the patient could become independent.

HISTOLOGIC DIAGNOSIS OF BREAST CARCINOMA WITH THE SILVERMAN NEEDLE BIOPSY

SIDNEY L. SALTZSTEIN, M.D.,* ST. LOUIS, MO.

(From the Division of Surgical Pathology, Washington University School of Medicine, Barnes Hospital, and the Barnard Free Skin and Cancer Hospital)

SILVERMAN needle biopsy of the breast has proved to be a simple and effective method of obtaining a positive histologic diagnosis of carcinoma under certain conditions. This method has been used with considerable success at this institution in a series of 160 breast lesions.

Aspiration biopsy, with the use of a syringe, needle, and smear technique, has been employed for many years as a relatively simple technique for the diagnosis of breast carcinoma.[13, 20, 21, 28, 30] Considerable success has been reported with this method, especially by the Memorial Hospital group. Correct diagnoses were established in 80 to 90 per cent of the lesions aspirated, depending on the experience of the operator.[13] Some authors believe that there are no ill effects resulting from this technique.[25] Even better results, 93 per cent of cancers detected by aspiration, have been reported.[28] Compared to histologic sectioning, the smear technique has an additional advantage of speed. The slides can be stained and observed immediately, without time-consuming processing.

Cytologic examination of breast secretions, by the Papanicolaou staining methods, also has been used with some success.[24, 26, 27]

Both of these methods have the inherent disadvantage that the diagnosis is based on cytology rather than histology. Therefore the pathologist must be experienced in cytologic interpretation if he is to establish the correct diagnosis.[1, 28] To obviate this difficulty, several authors have treated the aspirated fragments of tissue as a small tissue biopsy, imbedding them in paraffin and cutting histologic sections.[1, 5, 16, 17, 22] By this method, the architecture of the lesion can be studied. The Silverman needle[29] and other similar instruments[6, 8, 9, 16-18] have been used to obtain larger pieces of tissue.

MATERIAL AND METHODS

From January, 1952, through December, 1959, 171 needle biopsies have been performed on 160 breast lesions in Barnes Hospital, the Barnard Free Skin and Cancer Hospital, and the Washington University Clinics. Seventy-nine per cent were carried out on the ward service or in the clinics, and 21 per cent on private patients. The Silverman needle was used in the usual manner after skin preparation, local anesthesia, and small skin incision. The specimens were placed on a piece of paper towel or filter paper to prevent distortion, and fixed

Received for publication Feb. 4, 1960.

*Clinical Fellow of the American Cancer Society, Inc., 1959-1960.

in 10 per cent formalin. They were then dehydrated and imbedded in an automatic tissue processing machine, and sectioned at 6 μ. In general, 3 slides from various levels of the paraffin block were prepared, each slide containing several sections of the tissue. Hematoxylin and eosin stains were used. Diagnoses were based on the usual histologic criteria of breast lesions (Figs. 1-3).

All of the sections of the biopsy specimens and of any additional material in each case were reviewed by the author. In only one instance to be discussed below, was the diagnosis changed from that offered at the time of biopsy. The amount of tissue arbitrarily was considered inadequate for diagnosis if it did not extend the diameter of 1 low-power field (2 mm.), although in several instances a diagnosis of carcinoma could be made on this amount of tissue, or even less. The clinical records of the patients were also reviewed.

Fig. 1.—Silverman needle biopsy of carcinoma of the breast. (Hematoxylin and eosin; ×19; reduced ¼.)

RESULTS

Seventy-nine per cent of the needle biopsy specimens were interpreted as carcinoma, and 126 (91 per cent) of the 138 cases which eventually proved to be carcinoma were detected by needle biopsy (Table I). This corresponds quite favorably with the results (92 per cent) reported by Kaae.[18] According to figures previously published from Barnes Hospital and the Barnard Free Skin and Cancer Hospital,[2] 94 per cent of breast lesions which eventually were shown to be cancer were diagnosed histologically on frozen section.

TABLE I. RESULTS OF 160 BREAST NEEDLE BIOPSIES,* BARNES HOSPITAL AND ALLIED INSTITUTIONS, 1952 THROUGH 1959

DIAGNOSIS	DIAGNOSED ON NEEDLE BIOPSY	DIAGNOSES ON SUBSEQUENT TISSUE		
		CONFIRMED NEEDLE BIOPSY DIAGNOSIS	CHANGED DIAGNOSIS TO CARCINOMA	NO FURTHER TISSUE OBTAINED
Carcinoma	126	82	—	44
Not carcinoma	34	12	12	10
Intraductal papilloma	3	1	0	2
Chronic cystic disease	3	2	0	1
Fibroadenoma	1	1	0	0
Inflammation	4	3	0	1
None made	23	5	12	6

*The 11 breast lesions which were biopsied twice with the Silverman needle are counted in this table as single needle biopsies.

We consider a needle biopsy from which histologic sections are made to be the equivalent, except in size, of any other biopsy. Thus a histologic diagnosis of carcinoma established in this manner is as valid as one established by a larger

biopsy and, therefore, there are no false positive diagnoses. False positive results have been reported with the aspiration and smear technique.[28] Carcinoma was present in all of the 82 patients from whom additional material was

Fig. 2.

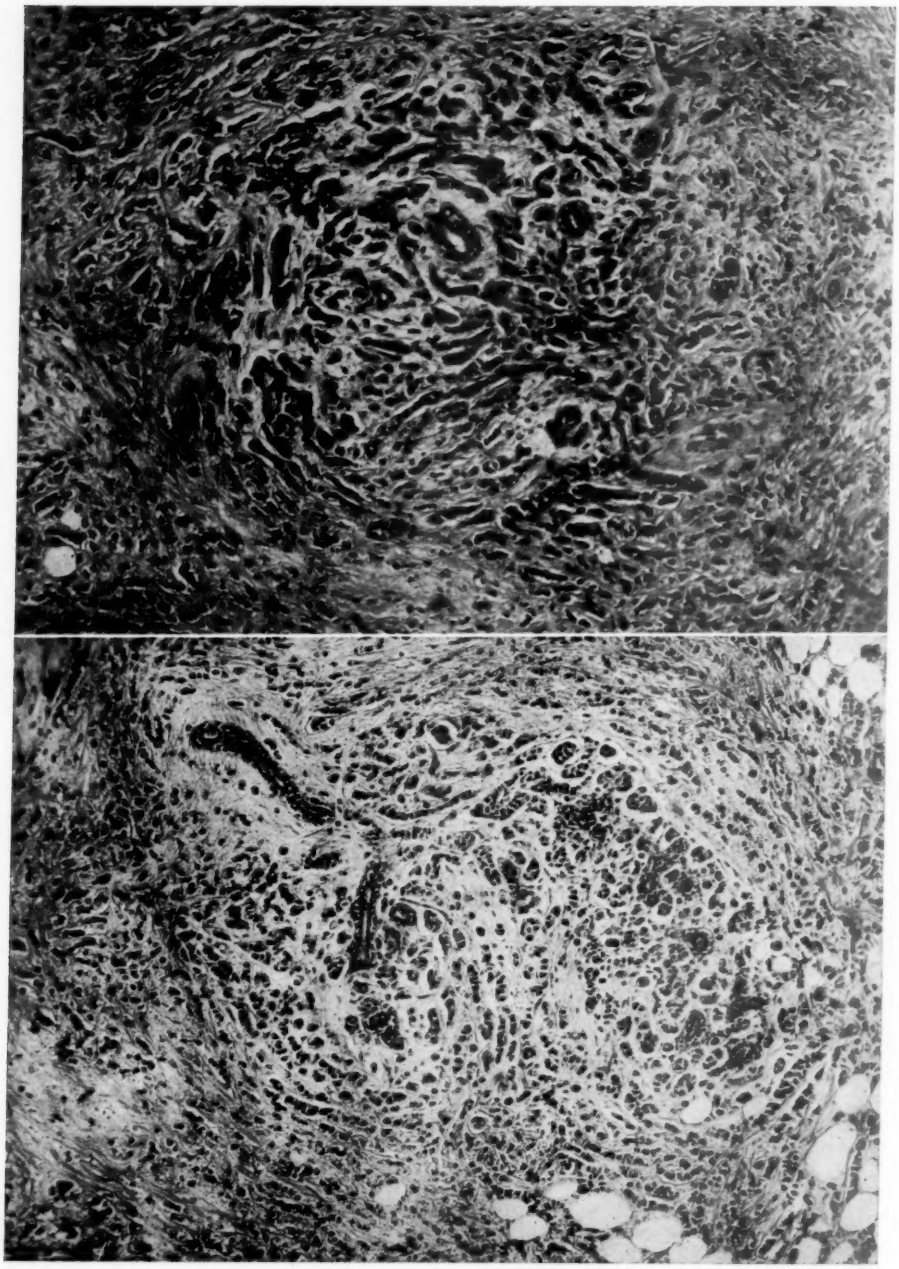


Fig. 3.

Fig. 2.—Silverman needle biopsy of carcinoma of the breast. Higher magnification of Fig. 1. (Hematoxylin and eosin; ×155; reduced ⅓.)

Fig. 3.—Carcinoma of the breast removed by radical mastectomy. Same case as Fig. 1 and Fig. 2. (Hematoxylin and eosin; ×130; reduced ⅓.)

obtained. Forty-nine of these underwent radical mastectomy, which represents 7 per cent of the 683 radical mastectomies performed in our institution during this same 8-year period. The 33 patients whose subsequent operative procedures were not radical mastectomies, and the 44 patients from whom no further tissue was obtained, represent those who were treated by irradiation, hormones, castration, or cytotoxic agents, or who refused further therapy.

Evaluation of a lesion in which the needle biopsy does not show carcinoma is more difficult. Twelve of the 34 patients (35 per cent) with a needle biopsy diagnosis other than carcinoma were later shown to have carcinoma. In all of these, the needle biopsy diagnosis was "none made." Seven specimens in the "none made" group were considered inadequate in amount by our criterion. Five of the patients in this group subsequently were found to have carcinoma. The 11 diagnoses of a specific benign lesion were subsequently confirmed either by removal of further tissue (7 cases) or by the benign clinical course of the patient (4 cases). A single needle biopsy of a breast lesion which does not show carcinoma is considered an indication for further investigation in spite of the success in diagnosis of the few specific benign lesions. The occasional instance in which a small focus of carcinoma in an otherwise benign lesion could be missed by the biopsy needle, as pointed out by Haagensen,[14] is the reason for further investigation of patients whose needle biopsies do not demonstrate carcinoma.

In 11 patients, 2 needle biopsies of a single breast lesion were performed. Two lesions showed carcinoma both times, and one of these was the only instance in which a needle biopsy diagnosis was changed on review. The interpretation at the time of the first biopsy was "none made," but on review was "carcinoma." The second needle biopsy, performed a few days later, showed carcinoma and virtually no time was lost in the treatment of the patient. Needle biopsies of 5 lesions did not contain carcinoma on the first attempt, but did on the second. In one case the first biopsy showed carcinoma, but the second, after radiotherapy was completed with clinical shrinkage of the tumor, did not show carcinoma. Both needle biopsy specimens were interpreted as "fat necrosis" in one case. Two lesions which subsequently were found to be carcinoma were interpreted as "none made" on both needle biopsies. Three of the 4 needle biopsies of these 2 lesions had to be considered inadequate in amount by our criterion. Thus, it would appear that with 2 adequate needle biopsies, one could expect to obtain a histologic diagnosis of carcinoma in even a higher percentage of cases than with a single needle biopsy.

Clinical factors which could have an effect on the success or failure of needle biopsies were also investigated. From each clinical record, the size of the lesion and a statement of the fixation were obtained from the report of the physical examination of the senior person who described these findings. Since the size was variously given in 1, 2, or 3 dimensions, the larger or largest dimension was used in the last 2 situations. These are presented in the tables as the "maximum dimensions." Where fixation was definitely described, the case is included in the appropriate "definite" columns. When an equivocal statement

TABLE II. COMPARISON OF CLINICAL FACTORS IN SUCCESSFUL AND UNSUCCESSFUL NEEDLE BIOPSIES OF BREAST CARCINOMA

	NO. OF CASES	CLINICAL SIZE OF LESION					FIXATION			
		AVERAGE MAXIMUM DIMENSION	SMALLER THAN 4 CM. (%)	4 CM. TO 6 CM. (%)	LARGER THAN 6 CM. (%)	"LARGE"* (%)	SKIN		DEEP	
							DEFINITE (%)	EQUIVOCAL (%)	DEFINITE (%)	EQUIVOCAL (%)
Successful biopsies (diagnosis of carcinoma made on needle biopsy)	126	6.3 cm.	13	42	32	13	73	11	28	3
Unsuccessful biopsies (diagnosis of carcinoma made on subsequent material, not on needle biopsy)	12	4.6 cm.	25	67	8	0	58	8	25	0

*These tumors are described in the clinical records as "involving most of the breast," "one-half of the breast," and so forth, without specific dimensions stated.

was made, or when only slight dimpling or retraction of the skin was described, the case is entered in the "equivocal" groups. All of these data were not available in every case.

The effect of the clinical factors on the success of establishing the diagnosis by needle biopsy is apparent in Table II. The difference in average maximum dimensions is statistically significant ($t_{122} = 2.7$; $p = 0.01$), as is the difference in number of lesions 6 cm. in diameter or smaller ($\chi^2 = 6.23$; $p = 0.015$). Definite fixation of the tumor to the skin probably has some bearing on a successful needle biopsy, although the difference in the 2 percentages is not statistically significant ($\chi^2 = 1.13$; $p = 0.25$). Deep fixation is equally frequent in both groups. The 101 ward cases and the 29 private cases included in this table are comparable in size of the lesions and presence or absence of fixation. The effect of the size of the lesion on the success of detecting a breast carcinoma is tabulated in Table III, and the experience of Barnes Hospital and Allied Institutions is compared with that reported by Kaee.[18] Here again, the larger the tumor, the greater the success in establishing the diagnosis by needle biopsy. Kaae's results are similar, but slightly better in the smaller lesions. Our ward and private series show almost identical percentages of success.

TABLE III. EFFECT OF CLINICAL SIZE OF BREAST CARCINOMA ON SUCCESSFUL DIAGNOSIS BY SILVERMAN NEEDLE BIOPSY

CLINICAL SIZE OF LESION (MAXIMUM DIMENSION)	PERCENTAGE OF LESIONS EVENTUALLY PROVED TO BE CARCINOMA DIAGNOSED BY NEEDLE BIOPSY			
	BARNES HOSPITAL AND ALLIED INSTITUTIONS			
	WARD (%)	PRIVATE (%)	ENTIRE SERIES (%)	KAAE[18] (%)
Less than 4 cm.	85	80	83	86.8
4 cm. to 6 cm.	86	85	86	93.6
Greater than 6 cm.	97	100	97	95.8
"Large"*	100	100	100	—
Total	91	90	91	92

*These tumors are described in the clinical records as "involving most of the breast," "one-half of the breast," and so forth, without specific dimensions stated.

No complications of the needle biopsy were described in any of our patients, except for one asthma attack apparently provoked by the biopsy.

DISCUSSION

This collection of 160 biopsies of breast lesions can hardly be considered a random sample of breast lesions. Eighty-six per cent of the lesions eventually proved to be carcinoma. On the whole, the tumors are large (average maximum dimension 5.7 cm.) and the malignant ones may be considered advanced in the course of the disease (75 per cent with definite skin fixation).

Nevertheless, the Silverman needle has a definite place in the management of carcinoma of the breast. Because it does not require the elaborate procedure or entail the slight anesthetic risk of a formal surgical biopsy, it can be carried out in the office or clinic so that when the patient is admitted to the hospital for definitive treatment the histologic diagnosis is already established. Similarly, a patient might be more readily willing to undergo a needle biopsy than an incisional biopsy, and biopsy earlier in the course of the disease might be accomplished.

While modern anesthesia has made surgery possible even for the "poor-risk" patient, a saving of anesthesia time can only be to the benefit of the patient. Frozen section, even when most skillfully made, requires an interval of time to accomplish. While the frozen section is being performed or after the pathologist's report is received by the surgeon, the patient is reprepared and redraped, and the surgical team changes gloves and gowns before proceeding with the radical mastectomy. If the histologic diagnosis can be established conveniently prior to surgery, all of this anesthesia time can be saved.[13] To quantitate the additional anesthesia time required for radical mastectomy when a frozen section is performed, the anesthesia records of the 76 radical mastectomies performed in Barnes Hospital during 1958 were reviewed. These operations took an average of 27 minutes longer when a frozen section was performed than when it was not performed (23 minutes on the ward service and 32 minutes on the private service).

The Silverman needle biopsy is considered to be less traumatizing to the tumor than incisional biopsy. A smaller opening into the confines of the tumor is made and less tumor is cut across than with a surgical incision. As a corollary to this, the needle biopsy should be performed as atraumatically as possible by one experienced in this technique, and multiple "stabs" at the tumor should be avoided. There will be less spillage of tumor-contaminated blood and tissue fluid with a needle biopsy performed prior to operation than with incisional biopsy and frozen section. All of these factors should lessen the risk of implantation and metastasis.

The needle biopsy is of extreme value after patients have been treated by nonsurgical means (irradiation, hormones, and others). In such instances the simplicity alone of the method recommends it. Similarly we have found needle biopsy very useful in establishing a histologic diagnosis of the primary lesion in disseminated breast cancer.

Some authors[22] also are of the opinion that the chance of infection is less with needle biopsy than with incisional biopsy. Others[5, 20] believe that there is less scarring after a needle biopsy, and hence less confusion in later operative or radiologic procedures.

At times, frozen section diagnosis has been carried out in Barnes Hospital on breast needle biopsy material, rather than on incisional biopsy tissue. While a diagnosis can be made in this manner, the frozen section on such material is technically difficult.

However, the needle biopsy is not without its drawbacks. Implantation of tumor along the needle tract is a possibility[4, 5, 13] and has been reported with needle biopsies of other organs.[3, 10-12, 23] Implantation of breast carcinoma has not been reported, probably because the needle tract is removed in surgically treated patients. The needle biopsy should be so planned that the entire needle tract will be excised with the radical mastectomy specimen if the patient is to be treated by operation.[5, 13] Fungation of the tumor, even with a surgical biopsy, in patients treated by irradiation is not a problem.[15, 20]

Possible dissemination of the tumor by the needle biopsy is another frequent objection to this technique. However, the work of Kaee[19] and of Robbins and his associates[25] show that no risk of this can be demonstrated.

One senior surgeon[7] objects to needle biopsy, especially in clinic patients who are seen by a great number of clinicians, because it obscures physical findings. The edema and inflammation, however minimal, will tend to confuse subsequent observers when they attempt to determine operability. This is not considered to be sufficient reason to discourage the use of the needle biopsy.

This series demonstrates that the Silverman needle can be used successfully in the biopsy of larger breast lesions. Before these results can be extrapolated to include smaller lesions with the goal of replacing formal surgical biopsy or frozen section, a controlled series of smaller lesions with biopsies by the various techniques must be studied.

SUMMARY

Silverman needle biopsy offers a simple and effective method of obtaining a tissue diagnosis of carcinoma of the breast. When patients with advanced lesions are to be treated with radiotherapy, needle biopsy saves time and expense for the patient, and provides a tissue diagnosis for the radiotherapist. Operating time is saved when patients are treated surgically. The reduced trauma and spillage of tumor associated with the needle biopsy compared to incisional biopsy should allow less chance of implantation and spread of the tumor.

Needle biopsy is of less value in smaller lesions. If the needle biopsy does not show carcinoma in a lesion which is clinically thought possibly to be carcinoma, surgical biopsy should be performed. In no case could any delay in therapy be ascribed to a false negative needle biopsy.

REFERENCES

1. Ackerman, L. V.: Surgical Pathology, ed. 2, St. Louis, 1959, The C. V. Mosby Company.
2. Ackerman, L. V., and Ramirez, G. A.: The Indication and Limitations of Frozen Section Diagnosis, Brit. J. Surg. **46**: 336-350, 1959.
3. Ackerman, L. V., and Wheat, M. W.: The Implantation of Cancer—An Avoidable Surgical Risk? SURGERY **37**: 341-355, 1955.
4. Allen, J. G.: In Allen, J. G., Harkins, H. N., Moyer, C. A., and Rhoads, J. E.: Surgery—Principles and Practice, Philadelphia, 1957, J. B. Lippincott Company, chap. 25, pp. 501-535.
5. Bernhard, W. G., Keim, W. F., Grubin, H., Wanner, H., and Sewekow, G.: Aspiration Biopsy in Lesions of Head and Neck, Am. J. Clin. Path. **26**: 1022-1030, 1956.
6. Biegeleisen, H. I.: New Biopsy Needle, Am. J. Surg. **94**: 678-682, 1957.
7. Butcher, H. B., Jr.: Personal communication.
8. Christiansen, H.: An Aspiration Trepan for Tissue Biopsy, Acta radiol. **21**: 349-351, 1940.
9. Christiansen, H.: Biopsy With Rotating Trephine for the Purpose of Histologic Diagnosis, Acta Radiol. **23**: 147-150, 1942.
10. Crile, G., Jr., and Vickery, A. L.: Special Uses of the Silverman Biopsy in Office Practice and at Operation, Am. J. Surg. **83**: 83-85, 1952.
11. Clarke, B. G., Leadbetter, W. F., and Campbell, J. S.: Implantation of Cancer of the Prostate in Site of Perineal Needle Biopsy, J. Urol. **70**: 937-939, 1953.
12. Dolley, F. S., and Jones, J. C.: Lobectomy and Pneumonectomy for Lung Suppuration and Malignancy, Lancet **59**: 268-278, 1939.
13. Godwin, G. T.: Aspiration Biopsy: Applications and Technique, Ann. New York Acad. Sc. **63**: 1348-1373, 1956.

14. Haagensen, C. D.: Diseases of the Breast, Philadelphia, 1956, W. B. Saunders Company, chap. 4, pp. 74-155.
15. Haagensen, C. D.: Surgical Biopsy *in* Pack, G. T., and Livingston, E. M.: Treatment of Cancer and Allied Diseases, New York, 1940, Paul D. Hoeber, Inc., vol. I, chap. 4.
16. Hoffman, W. J.: New Technique and Instrument for Obtaining Biopsy Specimens, Am. J. Cancer **15:** 212-220, 1931.
17. Hoffman, W. J.: Punch Biopsy in Tumor Diagnosis *in* Pack, G. T., and Livingston, E. M.: Treatment of Cancer and Allied Diseases, New York, 1940, Paul D. Hoeber, Inc., vol. 1, chap. 5.
18. Kaae, S.: Trephine Biopsy, Especially in Breast Cancer, Acta radiol. **30:** 427-434, 1948.
19. Kaae, S.: The Risk Involved by Biopsy in Breast Cancer, Acta radiol. **37:** 469-478, 1952.
20. Martin, H. E., and Stewart, F. W.: The Advantages and Limitations of Aspiration Biopsy, Am. J. Roentgenol. **35:** 245-247, 1936.
21. Martin, H. E., and Ellis, E. B.: Aspiration Biopsy *in* Pack, G. T., and Ellis, E. M.: Treatment of Cancer and Allied Diseases, New York, 1940, Paul G. Hoeber, Inc., vol. I, chap. 6.
22. Meatheringham, R. E., and Ackerman, L. V.: Aspiration Biopsy of Lymph Nodes, Surg. Gynec. & Obst. **84:** 1071-1076, 1949.
23. Ochsner, A., Dixon, J. L., and De Bakey, M.: Primary Bronchogenic Carcinoma, Dis. Chest **11:** 97-127, 1945.
24. Papanicolaou, G. N., Bader, G. M., Holmquist, D. G., and Falk, E. A.: Cytologic Evaluation of Breast Secretions, Ann. New York Acad. Sc. **63:** 1409-1421, 1956.
25. Robbins, G. F., Brothers, J. H., Eberhart, W. F., and Quan, S.: Is Aspiration Biopsy of Breast Cancer Dangerous to the Patient? Cancer **7:** 774-778, 1954.
26. Saphir, O.: Cytological Examination of Breast Secretions, Am. J. Clin. Path. **20:** 1001-1010, 1950.
27. Saphir, O.: Early Diagnosis of Breast Lesions, J. A. M. A. **150:** 859-861, 1952.
28. Smith, I. H., Fisher, J. H., Lott, J. S., and Thomson, D. H.: The Cytological Diagnosis of Solid Tumors by Small Needle Aspiration and Its Influence on Cancer Clinical Practice, Canad. M. A. J. **80:** 855-861, 1959.
29. Silverman, I.: A New Biopsy Needle, Am. J. Surg. **40:** 671-672, 1938.
30. Stewart, F. W.: The Diagnosis of Tumors by Aspiration, Am. J. Path. **9:** 801-812, 1933.

TUMORS OF THE NECK

JOHN E. SKANDALAKIS, M.D., STEPHEN W. GRAY, PH.D., NICK C. TAKAKIS, M.D., JOHN T. GODWIN, M.D., AND DAVID HENRY POER, M.D.,
ATLANTA, GA.

(From the Departments of Surgery and Pathology, The Piedmont Hospital, St. Joseph's Infirmary, and Grady Memorial Hospital, and The Department of Anatomy, Emory University)

IT IS probable that there is a greater variety of diseases to be found in the neck than any other area of similar size in the body. They range from the common pathologic changes in the thyroid gland to hemangioendothelioma of the submaxillary gland, of which only 1 case has been reported (Jackson[12]). Because of the architecture of the neck, packed with structures passing from head to thorax, a great number of these diseases become manifest as palpable masses or visible swellings. Neoplasms, inflammations, cysts, functional hypertension, and congenital anomalies stimulate the interest of the internist, the surgeon, the radiologist, and the pathologist, as well as the anatomist and the embryologist. Differential diagnosis alone has been the theme of many papers in the last decade.

The records of a large charity hospital (Grady Memorial Hospital) and 2 private hospitals (St. Joseph's Infirmary and The Piedmont Hospital) were examined for the 5 years 1954 to 1958. There was a total of 248,690 admissions (exclusive of newborn infants). Of these, 73,196 (29.4 per cent) were surgical admissions. Among the latter, there were 3,027 (4.13 per cent) patients who had swelling in the neck. This is 1.22 per cent of total admissions and is less than the 3.9 per cent reported by Slaughter and associates.[24] Their figures seem to be based on all patients admitted with neck mass present, while our figures represent surgical admissions only.

Of the 3,027 patients with neck masses, 1,411 had masses of primary thyroid origin, leaving 1,616 or 53.4 per cent with diseases of other origin, including metastases from the thyroid gland (Table I).

TABLE I. INCIDENCE OF CERVICAL TUMORS AMONG 248,690 ADMISSIONS, 1954 TO 1958

	NO. OF CASES	PERCENTAGE
Surgical admissions	73,196	
Neck masses found	3,027	1.22
Thyroid	1,411	46.6
Nonthyroid	1,616	53.4
Total	3,027	100.0

Several classifications of these cervical swellings can be made. Marshall and Uram[17] have organized them on an "anatomicopathologic" basis, providing room in 8 major, and a number of minor, categories for almost all possible lesions of the region. We have chosen a modification of the simpler classification

Received for publication Feb. 3, 1960.

presented by Slaughter and associates,[24] dividing our cases as (1) neoplastic, (2) inflammatory, and (3) congenital and other masses. Table II shows the relative incidence of these types.

TABLE II. NONTHYROID NECK MASSES*

	NO. OF CASES	PERCENTAGE
Neoplastic	1,370	84.8
Inflammatory	52	3.2
Congenital and others	194	12.0
Total nonthyroid masses	1,616	100.0

*Includes metastases from thyroid.

As could be expected, the inflammatory swellings are the fewest. Increasing use of antibiotics has played an important part in reducing the number of these lesions requiring operation. Unless bacteria develop resistant strains faster than research develops new antibiotics, we should shortly see the necessity for such operations eliminated. It is with the neoplastic swellings that the surgeon must now be most frequently concerned.

Neoplastic Masses.—Of the 1,370 cases, 1,061 or 77.5 per cent were malignant. Of the latter, 900 or 84.8 per cent were metastatic from other regions of the body. The remainder were primary to the neck (Table III).

TABLE III. NEOPLASTIC NECK MASSES

	NO. OF CASES	PERCENTAGES
Benign		
Salivary gland	165	53.4
Parathyroid adenoma	11	3.6
Others	133	43.0
Total	309	22.55
Malignant		
Primary	161	15.2
Metastatic	900	84.8
Total	1,061	77.45
Total (benign and malignant)	1,370	100.00

A metastasis requires search for a primary lesion which may frequently be small, obscure, and asymptomatic. In our series, 769 or 85.4 per cent of the metastatic tumors had their origin above the clavicle, while 131 of them had their primary sites below it (Table IV). This is a slightly greater frequency of supraclavicular origin than in Slaughter's[24] Illinois cases (79.4 per cent) and a lesser frequency than in Hendrick's[10] San Antonio series (93.2 per cent). Martin and Morfit[18] described cervical metastasis as the first symptom in 152 cases (93.3 per cent) from supraclavicular primary sites and 11 (6.8 per cent) from infraclavicular sites. In 3,888 cases of cancer, 210 (5.4 per cent) produced their first symptoms as metastatic cervical tumors. Table IV shows the locations in our series. Fig. 1 shows diagrammatically the relationship between cancer and location, emphasizing the most frequently found circumstances.

Inflammatory Masses.—Among the inflammatory group, constituting 6.2 per cent of cervical swelling, all but 2 were acute. These latter were tuberculous (Table V).

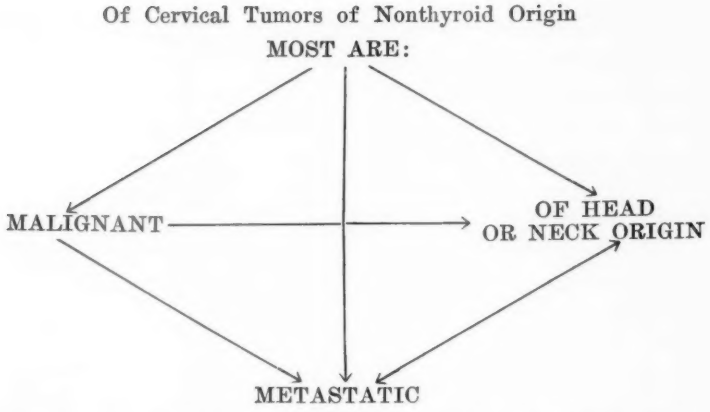


Fig. 1.—The most common circumstances found in connection with nonthyroid tumors of the neck.

TABLE IV. LOCATION OF MALIGNANT TUMORS

	NO. OF CASES	PERCENTAGES
Primary		
Major salivary glands	62	38.5
Malignant lymphoma	99	61.5
Total	161	15.2
Metastatic		
Primary site		
Above clavicle	769	85.4
Below clavicle	131	14.6
Total	900	84.8
Totals	1,061	100.0

TABLE V. INFLAMMATORY LESIONS

	NO. OF CASES	PERCENTAGES
Acute		
Simple lymphadenitis	47	
Lymphadenitis with abscess	3	
Total	50	96.15
Chronic		
Tuberculous lymphadenitis	2	
Total	2	3.85
Total (acute and chronic)	52	100.00

Congenital and Other Masses.—The third major group of cervical swellings are due neither to inflammation nor to neoplasm. There were 194 such swellings (Table VI). The larger number, 58.3 per cent, were those due to congenital defects resulting from failure of the embryonic pharynx to achieve entirely its mature development. Most frequent among these defects were cysts of the thyroglossal duct. They account for 72.5 per cent of our 113 congenital lesions. Various branchiogenic anomalies account for another 23.0 per cent. These tumors tend to manifest themselves early in life.

There has been some question as to whether cystic hygroma belongs to this group or to that of the neoplastic diseases. Slaughter and co-workers[24] include them in their neoplastic group, while Hendrick[10] and Marshall and Uram[17] do not. Their embryonic origin as sequestrations from developing lymph channels

is clear,[23] however, and they are as certainly congenital anomalies as are branchial or thyroglossal duct cysts. They are not limited to the neck.[28] They enlarge, become invasive, and frequently become infected, requiring surgical removal. In the present series they constitute 2.6 per cent of congenital and other masses (Table VI).

TABLE VI. CONGENITAL AND OTHER LESIONS

	NO. OF CASES	PERCENTAGES
Congenital tumors		
Thyroglossal duct cysts	82	
Branchiogenic anomalies	26	
Cystic hygroma	5	
Total	113	58.25
Salivary duct stenosis	54	27.84
Other	27	13.91
Totals	194	100.0

Salivary gland swellings due to blockage of the duct were responsible for 27.8 per cent of this group of swellings. Ductal calculus was the chief cause. Of the total nonthyroid swellings, 3.2 per cent were the result of this condition.

The remaining 27 swellings in this group were due to diverse causes such as esophageal diverticula, myocele, hematomas, congenital hypertrophy of neck muscles, and others.

DIAGNOSIS AND DISCUSSION

A few years ago, the patient with a lump on the neck often presented a problem. The work of Ward and Hendrick,[26] Hendrick,[10] Jackson,[12] and Marshall and Uram[17] has helped orient the general practitioner as well as the surgeon with respect to the clinical entities which may be encountered.

Certain diagnostic aids are available to the clinician as he observes masses in the neck. These signs are not definitive, but they are indicative. Of the neoplastic diseases, 75 per cent will occur in males. We do not know why. The inflammatory and congenital diseases will be equally distributed between the sexes.

A careful study of the patient's history will aid in diagnosis in many instances. The age of the patient and the time of onset are important. Hendrick's[10] statement is applicable: "Tumors and cysts which have been present for years are usually congenital in origin in contrast to those present for a period of months which are more probably neoplastic, and those present for a period of days, inflammatory. Pain early signifies, in most instances, an inflammation; when developing late it represents a neoplasm. . . ."

In the present series, the average age of the patients with neoplastic diseases was 60.9 years; with the inflammatory, 13.5 years; with the congenital, 7.9 years. The lump due to neoplastic disease has been observed by the patient over a period of *months* (7.3 is the average). The inflammatory swelling has been present for a matter of *days* (a week is the average). The congenital swelling has been present for *years,* usually since birth. This last is almost always painless, whereas the inflammatory tumor produces pain early and therefore is discovered early. The neoplastic tumor, although of long standing,

begins to produce discomfort only in the later stages. It will be convenient to remember these time intervals as a "rule of 7"—inflammatory tumors have usually existed for 7 days, neoplasms for 7 months, and tumors of embryonic origin have been present for 7 years before causing trouble. Table VII illustrates these relationships.

TABLE VII. DIFFERENCE IN INCIDENCE OF TYPES TO CERVICAL LESIONS

	SEX		AVERAGE AGE (YR.)	PERIOD MASS PRESENT	SYMPTOMS (PAIN)
	M	F			
Neoplasms	75%	25%	60.9	7.3 months	Late
Inflammatory	Equal		13.5	7 days	Early
Congenital	Equal		7.9	7.9 years	None*

*Unless inflamed.

As do all such generalizations, these statements represent only the most probable situation. To follow them blindly would all too frequently result in diagnostic failure. Martin and Morfit[18] have warned against one pitfall: "It is a common experience in cancer clinics that patients with histologically proved cervical metastases from demonstrable primary lesions will state that the cervical tumor, 3 to 5 centimeters in diameter, developed within a few hours. As a matter of general observational knowledge, it must take weeks or even months for a metastatic tumor to reach such a size."*

Thus, if the tumor is said to be of long standing, the patient should probably be believed. If it is said to be of recent growth, confirmatory evidence should be sought before an uncritical diagnosis of inflammatory lesion is made.

Specific diagnosis of neck tumors requires microscopic confirmation, preferably prior to definitive therapy. Material for diagnosis may be obtained by a variety of methods such as incisional, excisional, or aspiration biopsy.

Because of the ease and simplicity of aspiration biopsy,[7] this procedure is recommended as the initial step. Where diagnosis can be made from aspirated material, the distortion from incisional or excisional biopsy is avoided. This is important because the neck, once entered, presents a more difficult operative site for future exploration.

So far as is known, aspiration offers no greater hazard to the patient than do other methods of biopsy, and appears to offer less, because fewer vessels are usually disturbed. As with any other procedure, if a diagnosis is not forthcoming, another method such as excision may be employed. Spread of a tumor along the needle track has occasionally been reported as it has along paracentesis tracks, sutures, and surgical incisions. Such rare happenings cannot reasonably be held to contraindicate the use of these procedures. The needle track can and should be excised if subsequent surgical procedures are employed.

Aspiration biopsy is of the greatest assistance with metastatic lesions and is less useful in thyroid tumors. Fig. 2 shows malignant cells of a squamous cell carcinoma metastatic to a lymph node of the neck obtained by aspiration biopsy. Fig. 3 shows a similar metastasis from a blood clot aspirated from a lymph node of the neck.

*By permission of Surgery, Gynecology, and Obstetrics.

Certain characteristics of lesions of specific types may be examined in detail.

Congenital Lesions.—

Thyroglossal duct: Cysts and fistulas are midline lesions of congenital origin arising from epithelial remnants along the path of the embryonic thyroglossal duct. Such embryonic rests may lie anywhere between the foramen cecum at the base of the tongue and the suprasternal notch. They may deviate slightly to the right or left. The consistency of the tumor is soft and cystic.

Fig. 2.

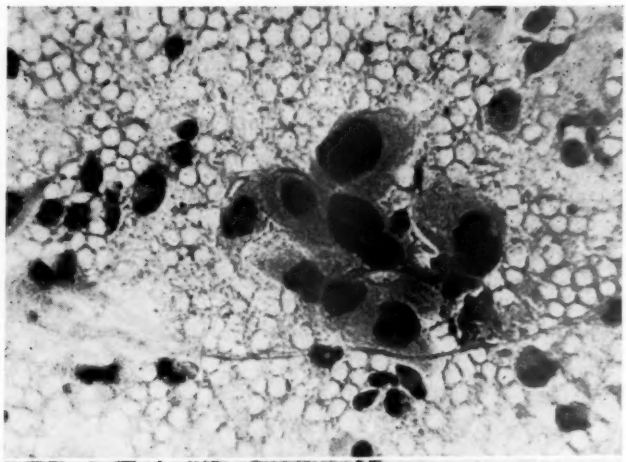


Fig. 3.

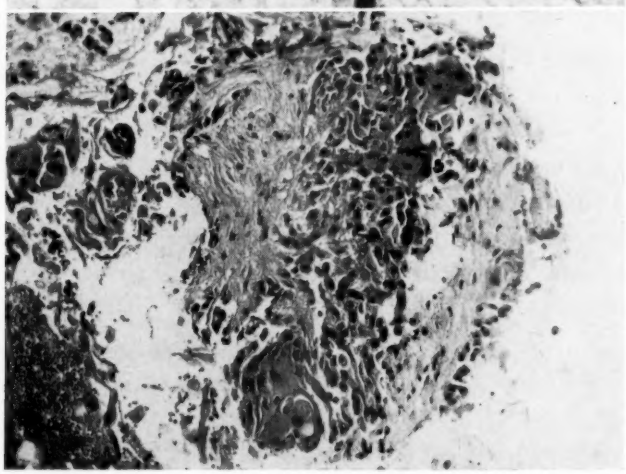


Fig. 2.—Photomicrograph of an aspiration biopsy smear showing malignant squamous cell carcinoma metastatic to a lymph node in the neck. (Hematoxylin and eosin; ×310; reduced ½.)

Fig. 3.—Photomicrograph of a sectioned blood clot showing malignant squamous cell carcinoma metastatic to a lymph node in the neck. (Hematoxylin and eosin; ×145; reduced ½.)

Beahrs[3] states that a cordlike structure representing the obliterated duct may frequently be palpated over the hyoid bone. Although they are most common in children, these tumors may be found in patients of any age. Sebaceous cysts, dermoid cysts, and the pyramidal lobe of the thyroid should be kept in mind as possible diagnoses.

Branchiogenic anomalies: These lesions differ from thyroglossal duct anomalies in their lateral position. Gross[9] found 17 per cent to be present on both sides. Most are derived from the second branchial cleft, although the first and third have been known to be involved. They are most commonly found at the mandibular angle, just anterior to the anterior border of the sternocleidomastoid muscle, but they may be found from the supraauricular region down to the level of the clavicle. They may become manifest at any age, and there is some evidence that their occurrence is familial. There is a question as to their potential malignancy. According to Ward and associates,[27] primary branchiogenic carcinoma may occur in 10 per cent of patients with branchial abnormalities. Neel and Pemberton[21] are of the opinion that such tumors are metastatic from an undiscovered primary lesion, probably in the nasopharynx. Martin, Morfit, and Ehrlich[19] have reviewed the literature of the disease and concluded that as a clinical entity "branchiogenic cancer is entirely hypothetical." Without denying the possibility of such lesions, they believe that there can be no histologic proof for its existence.

Cystic hygroma: This is a multilocular cystic lesion of embryonic lymph vessel origin. It may occupy the entire neck, and, though benign, it may be locally invasive. It nearly always appears in children in the first decade of life.

Benign Neoplastic Lesions.—Lipomas, sebaceous cysts, fibromas, neurofibromas, and other benign lesions do not present many real diagnostic problems. Sometimes the sebaceous cyst is found to be dermoid and the lipoma to be a fibroma. Since accurate diagnosis is not always possible, however, total surgical removal is always advisable.

Parathyroid adenoma will be diagnosed, not at the swelling in the neck, but by the systemic manifestations which it produces. No criteria or signs to assist in diagnosis are known for nonfunctional parathyroid tumors.

Malignant Neoplastic Lesions.—

Primary lymphoma: This lesion is extremely difficult to diagnose clinically and requires microscopic confirmation. We have classified our neoplasms of lymphoid tissue under the general rubric of "malignant lymphoma" with no attempt to subdivide them into giant follicle lymphoma, lymphosarcoma, Hodgkin's disease, or other classifications for this presentation. From a surgical standpoint, one or more unilateral or bilateral lymph nodes with enlargement, ovoid or not, with smooth or lobulated "nodular" surface, in upper or lower cervical region, usually the posterior triangle, are the characteristics of a malignant tumor. These tumors are generally radiosensitive but not radiocurable. Surgery plays only a diagnostic role in their management, though en bloc dissections are performed under suitable clinical conditions.

Metastatic: A nontender, discrete, hard, enlarged, movable or fixed lymph node in a patient over 50 years of age most probably represents a metastatic tumor. About one-half of the neck tumors developing in patients after the age of 50 are metastatic. Of malignant nonthyroid neck masses in our series, 84.8 per cent were metastatic, and only 15.2 per cent were primary. The primary site of the tumor was above the clavicle in 85 per cent of the cases and below in

15 per cent. Head and neck examination should be especially thorough. Upon failure to identify a primary tumor, thoracic and abdominal examination should be made. Left supraclavicular masses should suggest an intra-abdominal primary site.

Primary Salivary Gland Tumors.—Two hundred twenty-seven salivary gland tumors are included among our cases. Benign tumors account for almost three-fourths of them (72.7 per cent), and malignant tumors account for the remainder. These proportions are very similar to those of Kirklin and coworkers,[13] and to those in Foote and Frazell's[6] thorough monograph on these tumors. The latter authors found 69 per cent of 877 tumors to be benign, of which 82 per cent were classified as mixed. The benign tumors present as slowly growing, painless swellings, freely movable, with a firm cystic or nodular appearance.

The malignant tumors have a rapid rate of growth, and are usually fixed to skin, deep structure, or bone. They are hard, and in one-third of the cases, according to Ackerman,[1] facial nerve paralysis is present, often as an initial symptom.[6] Metastases to lymph nodes, lungs, and bone are frequent.

The potential malignancy of some salivary gland tumors is still controversial. Hendrick[10] reported 13 per cent to be primarily malignant and 10 per cent to be mixed tumors with malignant transformation. Both recurrence after incomplete ablation and aging of the tumor[4] have been held responsible for malignant transformation of these tumors.

Among the salivary glands, the parotid is the most frequent site of neoplasms. Surgeons and anatomists have long disagreed among themselves and between each other about the anatomy of the gland and its relation to the facial nerve. Table VIII shows the history of the descriptions of the parotid region.

TABLE VIII. HISTORY OF DESCRIPTIONS OF PAROTID REGION

AUTHOR	DATE	DESCRIPTION
Henle[11]	1873	Two unequal lobes divided by the facial nerve
Gregoire[8]	1912	Inverted V-shape with nerve in dividing connective tissue plane
McWhorter[16]	1917	H-shaped bilobar gland
Rouvière and Cordier[22]	1934	The deep lobe is double, a portion bridging to the superficial lobe above the facial nerve and a portion bridging between its main branches
Tortella[25]	1935	Noted the neglect of accurate description of course of parotid portion of facial nerve
McCormack, Cauldwell and Anson[14]	1945	Bilaminar with facial nerve in cleavage plane
Bailey[2]	1948	"Parotid sandwich"
McKenzie[15]	1948	Multiple small isthmi. Entwining of the gland into the trellis of the facial nerve
Davis and associates[5]	1956	Six types of distribution found among 350 dissections
Martin[20]	1952	Unilobular gland. Facial nerve branches are intimately enmeshed within glandular tissue
Winsten and Ward[29]	1956	

As late as 1901 Sir Frederick Trever stated that entire removal of the parotid gland was impossible.[5] As tumors of the parotid gland recur if excision is incomplete, it is of prime importance to understand the anatomy (and its possible variations) of the parotid gland and facial nerve. The best anatomic description of this region is probably that of Davis and his associates,[5] although the last word on this obscure area has probably not yet been published.

SUMMARY

1. More than 1,600 neck masses of nonthyroid origin were found over 5 years among 73,196 surgical admissions to 3 Atlanta, Georgia, hospitals.

2. Of the nonthyroid neck masses, 85 per cent were of neoplastic origin, 3 per cent were inflammatory, and 12 per cent were of congenital origin.

3. Of the neoplastic masses, 77 per cent were malignant, and of these, 85 per cent were metastatic and 15 per cent were primary.

4. Some diagnostic indications are described with special reference to aspiration biopsy as a simple method of differential diagnosis.

5. The "rule of 7" with respect to the onset of the tumor is introduced, not as an absolute diagnostic finding, but as a convenient mnemonic for the clinician (7 days, inflammation; 7 months, neoplasm; 7 years, embryologic origin).

REFERENCES

1. Ackerman, L. V.: Surgical Pathology, St. Louis, 1953, The C. V. Mosby Company.
2. Bailey, H.: Treatment of Tumours of the Parotid Gland, With Special Reference to Surgical Parotidectomy, Brit. J. Surg. **28**: 337-346, 1941.
3. Beahrs, O. H.: Differential Diagnosis of Tumors of the Neck, Minnesota Med. **38**: 293, 1955.
4. Buxton, R. W., Maxwell, J. H., and Cooper, D. R.: Tumors of the Parotid Gland, Laryngoscope **59**: 565-594, 1949.
5. Davis, R. A., Anson, B. J., Budinger, J. M., and Kurth, L. E.: Surgical Anatomy of the Facial Nerve and Parotid Gland Based Upon a Study of 350 Cervicofacial Halves, Surg. Gynec. & Obst. **102**: 385-412, 1956.
6. Foote, F. W., Jr., and Frazell, E. L.: Tumors of the Major Salivary Glands, in Atlas of Tumor Pathology, Armed Forces Institute of Pathology, Washington, D. C., 1954, National Research Council, sec. IV, fascicle 11.
7. Godwin, J. T.: Aspiration Biopsy: Technique and Application, Ann. New York Acad. Sc. **63**: 1348-1373, 1956.
8. Gregoire, R.: Le Nerf facial et la parotide, J. Anat. & Physiol., Paris **48**: 437-447, 1912.
9. Gross, R. E.: The Surgery of Infancy and Childhood: Its Principles and Techniques, Philadelphia, 1953, W. B. Saunders Company.
10. Hendrick, J. W.: Differential Diagnosis of Neck Tumors, South. M. J. **45**: 1019-1027, 1952.
11. Henle, J.: Handbuch der systematischen Anatomie des Menschen, Braunschweig, 1873, F. Vieweg, bd. 2.
12. Jackson, A. S.: Unusual Tumors and Cysts of the Neck, A. M. A. Arch. Surg. **70**: 895-905, 1955.
13. Kirklin, J. W., McDonald, J. R., Harrington, S. W., and New, G. B.: Parotid Tumors: Histopathology, Clinical Behavior, and End Results, Surg. Gynec. & Obst. **92**: 721-733, 1951.
14. McCormack, L. J., Cauldwell, E. W., and Anson, B. J.: The Surgical Anatomy of the Facial Nerve With Special Reference to the Parotid Gland, Surg. Gynec. & Obst. **80**: 620-630, 1945.
15. McKenzie, J.: The Parotid Gland and Its Relationship to the Facial Nerve, J. Anat. **82**: 183-186, 1948.
16. McWhorter, G. L.: The Relations of the Superficial and Deep Lobes of the Parotid Gland to the Ducts and to the Facial Nerve, Anat. Record **12**: 149-154, 1917.
17. Marshall, S. F., and Uram, H.: Tumors of the Neck and Parotid Areas: Report of More Than 1000 Cases, Postgrad. Med. **22**: 330-342, 1957.

18. Martin, H., and Morfit, H. M.: Cervical Lymph Node Metastasis as the First Symptom of Cancer, Surg. Gynec. & Obst. **78**: 133-159, 1944.
19. Martin, H., Morfit, H. M., and Ehrlich, H.: The Case for Branchiogenic Cancer (Malignant Branchioma), Ann. Surg. **132**: 867-887, 1950.
20. Martin, H.: The Operative Removal of Tumors of the Parotid Salivary Gland, SURGERY **31**: 670-682, 1952.
21. Neel, H. B., and Pemberton, J. deJ.: Lateral Cervical (Branchial) Cysts and Fistulas, SURGERY **18**: 267-286, 1945.
22. Rouvière, H., and Cordier, G.: Sur le développement de la glande parotide et les connexions qui existent entre les deux lobes de cette glande, Ann. d'anat. path. **11**: 622-624, 1934.
23. Sabin, F. R.: *in* Keibel, F., and Mall, F. P.: Manual of Human Embryology, Philadelphia, 1912, J. B. Lippincott Company, vol. 2, p. 709.
24. Slaughter, D. P., Majarakis, J. D., and Southwick, H. W.: Clinical Evaluation of Swellings in the Neck, S. Clin. North America **36**: 3-9, 1956.
25. Tortella, E. P.: Le plexus parotidieu du facial, Ann. d'anat. path. **12**: 41-50, 1935.
26. Ward, G. E., and Hendrick, J. W.: Tumors of the Head and Neck, Baltimore, 1950, Williams & Wilkins Company.
27. Ward, G. E., Hendrick, J. W., and Chambers, R. G.: Branchiogenic Anomalies, West. J. Surg. **57**: 536-549, 1949.
28. Ward, G. E., Hendrick, J. W., and Chambers, R. G.: Cystic Hygroma of Neck, West J. Surg. **58**: 41-47, 1950.
29. Winsten, J., and Ward, G. E.: The Parotid Gland: An Anatomic Study, SURGERY **40**: 585-606, 1956.

CARCINOMA OF THE TONGUE: ITS BEHAVIOR AND MANAGEMENT

JOSEPH E. HAMILTON, M.D., WRENO M. HALL, M.D., AND
PHIL J. HARBRECHT, M.D., LOUISVILLE, KY.

(From the Department of Surgery, University of Louisville School of Medicine and the Louisville Veterans Administration Hospital)

ALTHOUGH this accessible cancer gives fair warning when still over 80 per cent curable, one-third of the patients reached this hospital with a condition advanced beyond salvage. The patient is not alone to blame. Too often the first doctor consulted, unfamiliar with this field and loath to mutilate the patient, has ordered radiation to the tongue and has ignored neck nodes. Despite his humane intentions, he may well have denied the victim his chance for cure.

MATERIAL

From the hospital's opening in April, 1946, through December, 1957, 39 patients with cancer of the tongue have been admitted, all men ranging in age from 27 to 94 years with the average 56.7 years. Eighteen lesions occupied the free tongue, 11 the base, and 10 both free tongue and base. Follow-up on all patients is complete and all but one have been personally observed by the senior author.

Previous Treatment.—Eleven patients had been previously treated elsewhere, chiefly by x-ray. In 6 the cancer was hopelessly advanced at the time of entry. A seventh refused treatment and only one has proved salvageable.

Possible Predisposing Factors.—Leukoplakia was diagnosed in 3 patients. Serologic determinations were positive in 5. Bad oral hygiene was found in 22 instances. Thirty-one of 32 patients about whom information was available smoked or chewed tobacco (1 case) and of these, 18 did so moderately to excessively.

Stage of Disease.—As is common in institutions of this kind, a large proportion of the cancers were advanced and neglected. Thirteen of the total 39 patients had incurable disease and in 8 of these, the cancer was advanced beyond even palliative treatment.* This proportion is close to the 32 per cent of cases for palliation only found by Perzik and co-workers at Los Angeles County Hospital.[7]

There were also 4 nondeterminate cases. Of these, 2 patients refused treatment and are dead of disease, 1 died of terminal tuberculosis 2 weeks after radio-

---

Published with permission of the Chief Medical Director, Department of Medicine and Surgery, Veterans Administration, who assumes no responsibility for the opinions expressed or the conclusions drawn by the authors.

Received for publication Feb. 1, 1960.

*No patient is abandoned as incurable unless it is obvious that the most radical combined procedure cannot remove the growth and metastases or that an extensive or midline base-of-tongue cancer with metastasis will involve a total or near total glossectomy and laryngectomy along with resection of mandible and neck. In addition, radiotherapy, if used, is regarded as palliative only.

therapy was started, and the fourth died of a vascular accident 5 months after a combined resection (but, according to his family, he was free of disease). The remaining 22 patients, or 56.4 per cent, received definitive treatment for cure.

Youth as an Unfavorable Factor.—This was mentioned by Perzik and associates[7] but was not borne out here. Two of the 3 patients under 40 years of age were cured and 40 per cent of patients under 50 years survived, as compared to 28 per cent of those over 50 years.

Cervical Metastases.—As shown in Table I, 22 or 63 per cent of patients had cervical metastasis upon entry and in 5 others (14 per cent), metastases developed later for a total of 77 per cent. Bilateral or crossed metastasis appeared in 4 patients with unilateral, treatable cancer. This is exclusive of 5 hopelessly advanced cases in which bilateral cervical involvement was no longer of clinical significance.

TABLE I. STATUS OF CERVICAL LYMPH NODES
(Data Available on 35 Patients)

	NO.	%
Positive at entry	22	63
Became positive later	5	14
Stayed negative	8	23
Crossed metastasis, homolateral side uninvolved	2	
Bilateral metastasis, primary unilateral and curable	2	11

Size of the Primary Lesion.—Size of lesion as related to survival is shown in Table II. It is striking that all but 1 of the surviving patients had cancers not exceeding 2.5 cm. in diameter. Within this small series, size greater than 2.5 cm. appears even more adverse to prognosis than metastasis to cervical nodes. Thus, 4 of 11 treatable patients with positive nodes were cured in contrast to the 1 patient with primary lesion exceeding 2.5 cm. in diameter.

TABLE II. SIZE OF LESION RELATED TO SURVIVAL
(39 Patients)

SIZE	NO.	LIVED	DIED
5 cm. or larger	13	0	13
3 to 4 cm.	9	1	8
2.5 cm. or smaller	17	8	9

Recurrent or New Cancer?—Five separate cancers appeared in 4 patients. Twice this occurred on the same side of the tongue, 3 or 4 cm. from the original lesions destroyed 12 and 40 months previously. To avoid any question, these were termed recurrences, though in the latter with 40 month control this seemed most unlikely. Were this counted a new cancer, the net cure rate for the series would be 43.5 per cent instead of the 41 per cent indicated below. The other 3 cancers appeared on the opposite side, once in the base of the tongue, twice in the tonsillar fossa. These seemed beyond doubt new tumors, possibly on the basis of "field cancerization." Southwick and co-workers[9] reported this independent tumor phenomenon in 11.2 per cent of their 783 oral cancers, and are of the opinion that these are new and not recurrent growths.

TREATMENT

Of this group of 39 patients, 9 are free of cancer. This is a 23 per cent arrest for all patients, but 41 per cent for the 22 determinate and curable patients. The duration of cure is 2 years and 4 months for 1 patient, 4 years for another, and the rest have been free of disease 5½ to 13 years.

Predominantly Surgical Treatment.—Surgical treatment was used predominantly in 17 of the 22 curable patients (Table III). One died of a stroke 5 months postoperatively, but was still free of disease at follow-up 2 months after operation and at death according to his family. This leaves 16 definitive cases. Neck dissection was performed in all and bilaterally in 3. The local lesion was treated by operation alone 12 times, by radiation alone twice, and by combined operation and radiation 3 times. One of these last 3, a grade 3 base-of-tongue cancer, had recurred after radiation but the patient has had a 9-year cure following hemiglossectomy and neck dissection. This was the only cure among the 5 in which x-ray participated and represents 20 per cent, whereas 8 (67 per cent) of those treated by operation alone survived.

TABLE III. SURGERY AS THE PREDOMINANT TREATMENT
(16 Patients)

	%
Mortality (27 operations on 17 patients)	0
Living and well 2⅓ to 13 years (8 patients)	50
Nodes positive (11 patients, 4 salvaged)	36
Nodes negative (5 patients, 4 salvaged)	80
Treatment of primary lesion	
Operation only (12 patients, 8 salvaged)	66.6
X-ray with or without operation (5 patients, 1 salvaged)	20

Discontinuous versus Combined Operations.—In 9 patients, treatment of the primary and of the cervical nodes was separately performed with success in 4 or 44 per cent. In the other 7 patients, tongue, jaw, and neck were resected in a combined operation (in one a pull-through operation was performed with preservation of the jaw) and of these, 4 or 50 per cent are free of disease. Such a small experience does not warrant any conclusions concerning the relative merits of the 2 procedures when appropriately used. As far as could be determined, the failures in the discontinuously treated group were not related to the method but to distant or recurrent cervical metastases. Smaller lesions, not invading the floor of the mouth and unassociated with gross cervical metastasis, are treated by separate procedures, the combined operation being used for larger lesions reaching the floor of the mouth and with obvious nodal involvement. Kremen[2] achieved a 65 per cent 3-year salvage using a combined operation and sparing the mandible where feasible. Regardless of whether discontinuous or in-continuity resection is used, we urge wide local excision including at least 1.5 cm. of normal tissue beyond tumor induration at the narrowest point. Hemiglossectomy should also include greater anteroposterior clearance than this. The neck dissection employed is complete and radical, as described by Hayes

Martin and co-workers,[4] with the reservation that we do not usually find it safe to spare the marginal mandibular nerve in its posterior course.

Indications for Neck Dissection.—This remains an unsettled problem. Hayes Martin and co-workers and others reserved neck dissection for clinically positive nodes. However, many surgeons, ourselves included, routinely dissect the homolateral neck. There is increasing evidence that the salvage of patients with tumor-invaded nodes is far greater when the invasion is still microscopic than when it has become clinically detectable. Roux-Berger and associates of the Curie Institute[8] reported a 27 per cent 5-year survival when cervical metastasis was microscopic and only 11 per cent when it was grossly apparent. Perzik and his group[7] obtained 3-year salvage in 37 per cent of microscopic and 16 per cent of clinical metastasis. Ogura and Bello,[6] writing on laryngeal cancer, reported 71 per cent arrest when nodes were microscopically positive and only 33 per cent when they were clinically positive. In the case of malignant melanoma, McCune and Letterman[5] found the 8-year cure of microscopic metastasis to be 43 per cent whereas it was zero for gross metastasis.

The 2 necks in the present series (Table IV) in which only microscopic cancer was found remained uninvolved, though 1 patient died a year later from contralateral metastasis recurring after therapeutic neck dissection. On the other hand there was recurrence after 6 of the 10 therapeutic dissections for gross metastasis. The survival following prophylactic dissection was 55.5 per cent and following therapeutic dissection 30 per cent.

TABLE IV. OUTCOME OF 19 NECK DISSECTIONS, 17 PATIENTS

	NO.	
Prophylactic dissections (2 microscopic metastases, both controlled)		9
Death from contralateral uncontrollable metastasis	3	
Death from recurrent primary cancer	1	
Patients cured	5 (55.5%)	
Therapeutic dissections (10 gross metastases confirmed, 4 controlled)		10
Death from cervical recurrence alone	3	
Death from cervical recurrence with recurrent primary cancer	2	
Death from cervical recurrence with distant metastasis	1	
Death from distant metastasis alone	1	
Patients cured	3 (30%)	

Whether or not to dissect the clinically uninvolved contralateral neck deserves some consideration. Lyall and Schetlin,[3] on the basis of a 67 per cent 5-year arrest in 18 patients with bilateral neck dissection, recommend it for all cases of cancer of the tongue whether the nodes show clinical evidence of involvement or not. Their 28 per cent incidence of bilateral metastasis would probably indicate such a procedure but this incidence is much higher than that found by most observers. As shown in Table I, there were 4 instances (11 per cent) of crossed or bilateral metastasis in the present series, in 2 of which the homolateral nodes remained negative. Furthermore, in 3 of the 4 cases recurrent

cancer developed after dissection and the patients died. A few more cases such as these might well convince us of the wisdom of routine bilateral prophylactic neck dissection.

Radiation Therapy.—Eleven patients underwent radiation therapy for cure (Table V) and it was the sole treatment for 6 of these. One patient has remained cancer-free 6 years. In the other 5, the cancer grew and metastasized

TABLE V. RADIATION THE PREDOMINANT LOCAL THERAPY

	NO.	
Total local lesions treated		21
Palliative treatment only		10
Curative attempts (success in 3 or 27%)		11
Radiation alone (one 6-year cure)	6	
Radiation plus surgery* (2 controlled)	5	

*Both patients with controlled primary cancer died of metastases in 11 months and 28 months. One postradiation recurrence was controlled by surgical excision.

beyond curability under treatment, the primary cancer never coming under control. X-ray combined with operation achieved local control in 2 patients, but both died later of metastasis. Until recently, radiation therapy has consisted in combined external radiation with the 250 kv. machine and interstitial radon or intraoral cone. This has not proved as effective as wide surgical excision. Since 1957, however, a few encouraging results have been achieved, especially in lesions of the base of the tongue, with external cobalt radiation together with the use of interstitial low intensity radium needles. In addition, several large, ulcerating lesions of the tongue and floor of mouth have been treated by subtherapeutic doses of x-ray or cobalt followed by a combined en bloc surgical excision, but it is too early to comment on these.

SUMMARY AND CONCLUSIONS

The inverse relation of cancer size to curability in this series is striking. In 8 of the 9 arrests, the primary lesion measured 2.5 cm. or less (the ninth being 3 cm.) despite the fact that in 4 of these the cervical nodes were positive.

The literature reveals that the salvage of patients whose cervical nodes are still only microscopically positive is over twice as great as it is for those with clinically obvious metastasis, which coincides with our own findings. This is especially important when we recall that cervical nodes, clinically negative, will prove histologically positive 17 per cent to 60 per cent of the time on the homolateral side[1-4] and 10 per cent to 28 per cent on the contralateral side.[3, 4] This, in our opinion, calls for homolateral neck dissection in all cases and an increasing consideration of bilateral dissection.

Results of, and indications for, combined and discontinuous resection as well as radiation therapy are presented.

Five separate intraoral cancers in 4 patients of this series are described.

The over-all salvage (2⅓ to 13 years) in this group of 39 patients, including nondeterminate and incurable cases, was 23 per cent but for the curable cases was 41 per cent. The arrests primarily by operation were 50 per cent, by x-ray 27 per cent.

REFERENCES

1. Beahrs, O. H., Devine, K. D., and Henson, S. W., Jr.: Treatment of Carcinoma of the Tongue; End Result on One Hundred Sixty-Eight Cases, A. M. A. Arch. Surg. 79: 399, 1959.
2. Kremen, A. J.: Results of Surgical Treatment of Cancer of the Tongue, SURGERY 39: 49, 1956.
3. Lyall, D., and Schetlin, C. F.: Cancer of the Tongue, Ann. Surg. 135: 489, 1952.
4. Martin, H., del Valle, B., Ehrlich, H., and Cahan, W. G.: Neck Dissection, Cancer 4: 441, 1951.
5. McCune, W. S., and Letterman, G. S.: Malignant Melanoma, Ten Year Results Following Excision and Regional Gland Resection, Ann. Surg. 141: 901, 1952.
6. Ogura, J. H., and Bello, J. A.: Laryngectomy and Radical Neck Dissection for Cancer of the Larynx, Laryngoscope 62: 1, 1952.
7. Perzik, S. L., Joergenson, E. J., Carter, R. P., and Deeb, P. H.: End-Results in Carcinoma of the Oral Cavity, A. M. A. Arch. Surg. 76: 677, 1958.
8. Roux-Berger, M. L. J., Band, and Courtial, M. J.: Cancer de la partie mobile de la langue, Le curage ganglionnaire prophylactique est-il justifié? Statistique de la Fondation Curie. Mém. Acad. chir. 75: 120, 1959.
9. Southwick, H. W., Otten, J. W., and Slaughter, D. P.: Cancer of the Tongue, SURGERY 43: 313, 1958.

TEMPORARY REGIONAL SURFACE COOLING AND LONG-TERM HEPARINIZATION IN THE THERAPY OF BURNS

GEORGE MARK BERBERIAN, M.D., PONTIAC, MICH.

(From Pontiac General Hospital)

FACTORS responsible for thermal injury produce different degrees of damage to the affected areas. Tissue damage is related to the physical properties (solid, fluid, gas, radiation), duration of application, and thermal gradient of the burning agent.

Tissue damage is primary in some areas and secondary in others, i.e., some cells are totally destroyed initially because of the intensity of the heat generated at the site of burn. Other cells are damaged later as a result of secondary vascular thrombosis in and about the burn area.

Little can be done to save the initially destroyed cells, but protective measures can be taken for cells in danger of secondary damage. In analyzing the elements of cellular damage in thermal injury in a secondary pattern, one encounters the following:

1. Tissue edema in and about the burn area impairs to a certain extent the vascular function of the area.
2. Hemoconcentration increases tendency to vascular thrombosis.
3. With impaired vascular channels in and about the burn area, cells with reversible damage are apt to suffer from anoxia unless the metabolic pattern is lowered temporarily.
4. Damaged cells have a tendency to liberate their fatty contents. Fat globules are found in the systemic circulation in severe burns and are probably responsible for some of the local and systemic manifestations.

Unless action is taken to avoid or minimize secondary tissue damage the burn area is apt to appear worse in the days following the initial injury.

CLINICAL DATA

Forty patients at Pontiac General Hospital with burns of 5 to 20 per cent of the body surface and mostly of second, deep second, and third degree were treated as follows:

When the patients arrived in the emergency room, ice cold towels (dipped in ice cold water and rinsed out) were applied to the burn areas and replaced as soon as they lost their coolness. These applications were administered for 5 hours and then continued only if the patient complained of pain. The late applications were confined to the painful areas. Once the patient felt comfortable cold towels were discontinued and all burn areas were exposed to the open air.

Prothrombin and bleeding times were checked when the patients arrived and they were given heparin immediately. Adults received 10 mg. intra-

Received for publication Jan. 11, 1960.

muscular heparin sodium every hour for 5 doses and later 25 mg. every 6 hours until all burns healed. Children received 5 or 10 mg. heparin every 2 hours for 3 times and later 10 mg. every 6 hours until complete healing occurred. Intensive active mobilization and constant elevation of burn areas were carried out from the time of admission.

No cleansing or débridement of burn tissues was attempted unless tissues were dangling. In the later phase of healing a very limited débridement was carried out occasionally to check the possibility of pus pockets or remove detached crusts. Under no circumstances was adherent burn tissue débrided. Penicillin, 1 million units, and streptomycin, 1 Gm., daily were used in burns of above 15 per cent of the body surface and in children who were uncooperative.

Burns treated by the above method had been caused by steam heat, boiling water, gasoline flames, burning paint, contact with hot stove, and other accidents.

STATISTICAL ANALYSIS AND RESULTS

1. Nineteen adults and 21 children were submitted to this regimen of therapy.

2. The majority of the patients, 36 out of 40, did not require any narcotic, barbiturate, or analgesic at any time.

3. Weeping of burn areas was minimal at all times.

4. The degree of edema in and about the burn area was minimal. Some areas not subjected to cooling in the same patient showed an increased tendency to blister.

5. No hemorrhages appeared in and about the burn areas. One patient lowered his leg inadvertently and was subject to minimal bleeding within a few blisters. Bleeding stopped promptly as soon as elevation of leg was reinstituted.

6. Deep second and third degree burns revealed a minimal tendency to contracture and scars were observed to be soft and glistening.

7. Gross infection of burns was occasional and it was the impression of the observer that the need for prophylactic antibiotic was not justified in the above-mentioned cases. Occasional minor infection occurred equally in treated and untreated cases. Local débridement controlled adequately all such episodes.

8. Two patients required skin grafts, both on the distal portion of the legs.

COMMENTS

Several factors are involved in the therapeutic approach described:

Temporary Regional Surface Cooling.—Prolonged, intensive cooling can be destructive to tissue. By using cold towels and replacing them when they no longer feel cool, one avoids this destructive property of cold. Local cooling may be helpful in the following ways: (1) the metabolic rate of the burn area is reduced by the application of cold. Thus anoxia is less likely to occur. (2) Cooling is discontinued as soon as the patient is comfortable because tissue healing is slowed down by cooling. Thus, once the tendency to edema and blistering is overcome there is no need for local cooling. (3) By reducing

edema, cooling will improve venous and lymphatic drainage. Many investigators have shown the phenomenon of platelet escape from the peripheral circulation during generalized surface cooling. A low platelet concentration in the burn area treated by cold might lessen the occurrence of thrombosis. (4) Finally, all inflammatory phenomena are less liable to occur under local cooling.

Long-term Heparinization.—Heparinization was instituted progressively which allowed detection of a tendency to bleeding in burn areas and treatment if bleeding occurred. The action of heparin is many fold: (a) heparinization reduces the tendency to sludging of red cells and thrombosis; (b) heparin is anti-inflammatory and seems helpful in the treatment of phlebitis (it is estimated that superficial veins in and about the burn area are often in a state of phlebitis); (c) heparin improves oxygen consumption in tissue cultures; (d) heparin performs a clearing function on fat globules liberated from burn areas.

Active Mobilization and Elevation.—Active mobilization and elevation assure adequate circulation to affected areas. During the cooling period, mobilization counteracts the possible dangers of extreme cooling.

We conclude that heparinization and cooling will assure a better chance for survival of cells which are not destroyed by the thermal episode in the initial stage.

SUMMARY

The course of primary tissue destruction in thermal injury is unalterable. For prevention or amelioration of secondary damage, early and short-term local cooling under systemic heparinization with active mobilization and elevation of affected areas proved to be most gratifying in the treatment of the 40 cases reported involving deep second or third degree burns of 5 to 20 per cent of the body surface.

Since the introduction of the present therapy (July, 1959), the author has not had occasion to see or treat a clinical burn involving over 20 per cent body surface and of at least second degree. Thus, at present, no clinical data are available concerning the use of cooling and heparinization in major burns.

The author has collected experimental data on dogs subjected to hot water submersion burns (80° C. for 30 seconds), covering 40 to 50 per cent of body surface. These experimental data will be incorporated in a later report on the clinical use of cooling and heparinization in the therapy of severe thermal injuries.

I would like to express my appreciation to the entire Attending Staff of Pontiac General Hospital for their cooperation in making this investigation possible.

REFERENCES

1. Brooks, J. W., Robinett, P., Largen, T. L., and Evans, E. I.: Standard Contact Burn: Method of Production and Observations on Blood Pressure Following Its Production in Dogs, Surg. Gynec. & Obst. **93**: 543-554, 1951.
2. Elman, R., Cox, W. M., Jr., Lischer, C. E., and Mueller, A. J.: Mortality in Severe Experimental Burns as Affected by Environmental Temperature, Proc. Soc. Exper. Biol. & Med. **51**: 350-351, 1942.
3. Elrod, R. P., McCleery, R. S., and Ball, C. O. T.: Experimental Study of Effect of Heparin on Survival Time Following Lethal Burns, Surg. Gynec. & Obst. **92**: 35-42, 1951.
4. Stone, H., and Martin, J. D.: S. Forum **9**: 58, 1958.

STRANGULATED OBTURATOR HERNIA

FRANK A. ROGERS, M.D., WHITTIER, CALIF.

OBTURATOR HERNIA is an uncommon but important hernia because of its potentially lethal consequences. Its significance increases when one realizes how infrequently it has been correctly diagnosed prior to laparotomy. In order to emphasize these features, a group of 12 cases of strangulated obturator hernia are summarized from the records of the Los Angeles County General Hospital. These cases represent the only series of patients from an institution.

Historically, this type of hernia was first described by the French. Original manuscripts indicate that Pierre Roland Arnaud de Ronsil deserves credit for the first report of this type in a presentation of 2 cases before the French Royal Academy of Science in Paris about 1724. His paper was not published nor was the second report of this type of hernia given by Duverney before the same Society a few years later. G. Arnaud de Ronsil, son of Pierre, further discussed obturator hernia 44 years after the original report by his father.[11]

Camper described the anatomy of oburator hernia in 1726, and de la Garrene added a second case report in the same year. By 1743 de Garengeot[11] had collected 6 cases and had added another of his own. Heuerman, Cloquet, and Lacroissant were also early observers.

Hilton described the first laparotomy for strangulated obturator hernia in 1848, but credit is given by Erichsen[8] to Henri Obre for the first case diagnosed with recovery after operation (1851). Colson (1863) and Godlee (1885) also published reports of operations on this unusual type of hernia.

Horine[17] reviewed the history of obturator hernia and carefully reviewed the literature in 1927. He found 258 cases up to 1924. By 1946 Watson[34] had found 442 cases which he used as a basis for a collective review. Articles by Rowe and Bynum[23] and by Harper and Holt[14] have currently summarized experiences with obturator hernia. The latter authors reported 2 cases of their own in 1956, bringing the total recorded cases to 463 since the first report by de Ronsil in 1724. This figure is probably only an approximation of the frequency of this hernia.

ANATOMIC DESCRIPTION AND RELATIONSHIPS

A knowledge of the anatomy of this hernia is important to the understanding of the origin and subsequent symptomatology of such a hernia. Such knowledge also will make clear the inadvisability of repair from the thigh. The obturator foramina are found bilaterally between the ischium and pubis on anterior lateral pelvic wall. The foramen is closed by a fairly tough membrane except in the upper portion where the obturator artery and nerve make their exit from the pelvis. The obturator nerve supplies the skin, fascia, and

Received for publication Jan. 18, 1960.

muscles over the medial part of the thigh. It gives a branch to the hip joint and the posterior part of the knee. The artery, usually a branch of the hypogastric, generally supplies the same area.

The deep groove in the superior portion of the membrane is converted into a canal or funiculus by a strong ligamentous portion of the pelvis by the obturator internus muscle and on the outside by the obturator externus. The canal is 2 to 3 cm. long and ends beneath the pectineus, adductor longus, and obturator externus muscles deep in the medial thigh.

The usual obturator canal opening measures about 1 cm. These openings tend to be oval and smaller in males than in females. They are more triangular in females. Usually the canals are occluded with preperitoneal fat. The fatty layer lies between the inner pelvic peritoneum and the underlying endopelvic fascia which covers the obturator internus muscle and enters the obturator canal to fuse with the tissue of the obturator membrane.

Studies by Singer, Leary, and Hofmeyer[26] have suggested that "pilot tags" of subperitoneal fat covered with connective tissue may constitute a prehernial condition. They found such fatty protrusions herniating into the obturator canals of 16 out of 44 bodies dissected. The average specimen age was about 70 and the female to male ratio was 3 to 1. Anson,[2] however, found no obturator hernia and only 6 instances (3 per cent) of preperitoneal protrusion in 365 dissected body halves.

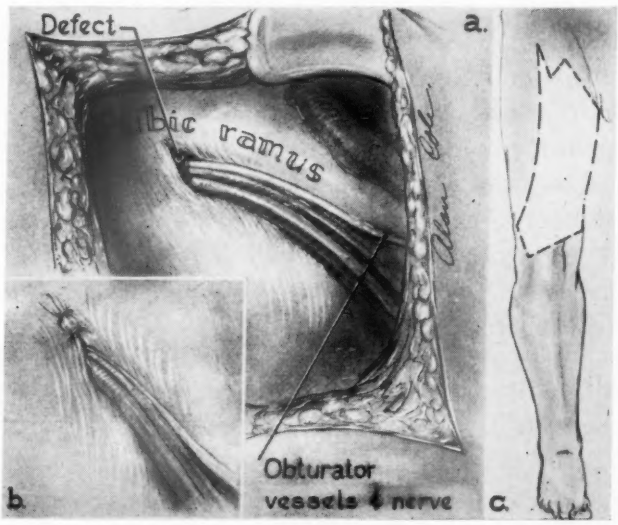


Fig. 1.—*A*, View of right obturator canal area from above. The nerve lies above the artery and companion vein. *B*, Simple interrupted suture closure will usually be sufficient to close the obturator defect after release of the hernia. *C*, Unshaded area of anteromedial thigh represents area of distribution of the obturator nerve and artery and is the area of the pain which occurs with obturator hernia (Howship-Romberg sign).

The relationship of the hernial sac to the vessels has been studied in detail and may vary in its location exterior to the pelvis. In its descent it may come to lie in the upper part of the thigh under the pectineus muscle in relation to the anterior branches of the obturator artery, or follow the inferior branches of the vessels to a slightly deeper location. The sac has also been described in a position behind the external obturator muscle. The neck of the hernia sac is medial to the artery and nerve as it passes through the fascial defect. The nerve lies just above the companion artery. The actual location of the sac in the thigh is of little significance as it is virtually impalpable.

The fascial defect in the great majority of strangulated hernias has been relatively small, as seen more clearly after reduction. One is amazed at the small, sometimes slit-like opening through which herniation can occur. The entrance to the canal is rather

rigid, being primarily fascia, and is somewhat unrelenting in its grasp once a portion of viscera has been incarcerated. Once incarcerated, strangulation of the sac contents is almost inevitable. The majority of obturator hernia sacs contain small bowel, and the Richter-type hernia is common. Five of the 12 cases reported here were of this type. Cases have been recorded in which the sac contained cecum, appendix, Fallopian tube, bladder, Meckel's diverticulum, and omentum. Fig. 1 illustrates the appearance of the right obturator canal from above. Nine of the 12 cases were right-sided obturator hernias. Three were left-sided hernias. This same incidence holds true for obturator hernia in general and correlates well with inguinal and femoral hernia where approximately a 2:1 ratio favoring the right side is noted.

The anatomic reasons for the predominance of females with obturator hernia (6:1 ratio females to males) are probably the normally larger obturator canal opening and, in many cases, the relaxation of the pelvic peritoneum due to repeated pregnancies. Most of the patients are elderly, usually in the seventh and eighth decades. Many are poorly nourished. Emaciation may reduce the effectiveness of the normally protective endopelvic preperitoneal fatty layer. Chronic pulmonary disease with cough and chronic constipation have been cited also as predisposing causes of this hernia. The majority of patients are over 50 years of age. The patients at the Los Angeles County General Hospital averaged over 75 years of age with the exception of the patient in Case 2, a 37-year-old female. Ten of the 12 cases reported were in females.

SYMPTOMS AND SIGNS

Clinically, strangulated obturator hernia produces the classical symptoms of small bowel obstruction. Cramping abdominal pain is noted first, followed by nausea and vomiting. Distention is usually not a prominent finding. The outstanding symptom characterizing this type of obstruction consists of pain or paresthesia felt down the anteromedial thigh to the knee with movement of hip or thigh. This has become known as the Howship-Romberg sign and is apparently produced by pressure on the obturator nerve from the hernia sac with its contents in the obturator canal. This type of pain, which follows the obturator nerve distribution, was described by Howship in 1840 and Romberg in 1845. Occasionally only paresthesias are noted by the patient. Rose[21] has recorded a case where sciatic pain was the principal symptom and a right obturator hernia was found at operation.

The vast majority of cases have not been diagnosed correctly before operation. In many instances this is because the diagnosis of strangulated obturator hernia was not considered. Only rarely, if ever, can a mass be palpated in the thigh. Pelvic or rectal examination may be suggestive of a laterally placed protuberance or tumor mass but in general has not been revealing. Two patients in the present series were suspected of having an obturator hernia and were specifically examined rectally and vaginally, but no mass was found. In another case, an unsuccessful attempt was made to feel the hernia through the vagina with the abdomen open and the hernia exposed from above. Some patients lack the symptoms of pain along the course of the obturator nerve. Others may lie with thigh flexed to partially relieve pressure on the nerve.

Recently several authors have published case reports where the diagnosis was correctly established preoperatively.[6, 14, 23, 29] In the present series the usual preoperative diagnosis was intestinal obstruction.

DIAGNOSIS

The differential diagnosis of strangulated obturator hernia is chiefly that of intestinal obstruction. The combination of acute small bowel obstruction with the Howship-Romberg sign is the most valuable aid in the diagnosis of this type of hernia. Several of the patients have previously had transient episodes of intestinal colic with associated thigh pain.[19, 27, 34] The colicky pain may be generalized across the mid-abdomen or more localized to the right or left lower abdomen. In a large number of instances the incarceration may be a Richter's type of hernia. This type of strangulated hernia should be considered in any older, thin female with signs and symptoms of intestinal obstruction who has not had previous abdominal surgery. The coexistence of a true Howship-Romberg sign in this symptom complex would be essentially pathognomonic of obturator hernia.

Two of the present group of 12 cases were elective repairs with no pain or acute symptoms. In 4 of the other cases a classical Howship-Romberg sign was described but was correctly interpreted in only 1 instance (Case 3). In 8 of the other 10 acute cases, mechanical small bowel obstruction was properly diagnosed prior to operation.

TREATMENT

Early surgery is the treatment for this type of strangulation intestinal obstruction. The incidence of gangrenous bowel is high. Several opinions exist regarding the proper approach for the release and repair of this hernia. A vertical exploratory laparotomy incision has been most frequently used in the past. If not placed too high it offers a perfectly adequate approach for release and repair of these hernias. Historically, the thigh or obturator approach was used by Anderson in 1900, while Milligan made use of an inguinal approach in 1919. Both of these incisions are entirely inadequate for proper exposure, since neither offers a clear view of the obturator defects or would allow a safe release of a tightly incarcerated loop of intestine or its subsequent resection. Obturator, inguinal, and abdominal approaches have been described by Wakeley.[32]

An excellent approach used enthusiastically by the author is the retropubic or Cheatle-Henry approach. This incision is placed in the suprapubic region and affords a direct view of either the right or left side of the public arch area. It is ideal for unilateral or bilateral repair of femoral and obturator defects and is adaptable as well for the repair of inguinal hernias. Its use for obturator hernia repair has been described also by Soothill.[27] If the diagnosis can be made before operation, a retropubic approach is advised. If gangrenous bowel is encountered, a release can be effected by the closest route possible and an opening in the peritoneal sac allows the operator immediate access to the abdominal cavity. The closure of bilateral obturator hernia defects, which occur in 1 out of every 15 cases (6.5 per cent), would be both possible and advantageous through this one incision. It is suggested that the apparently unaffected side be

TABLE I

CASE NO. YEAR	AGE RACE SEX	SYMPTOMS, DURATION, FINDINGS	HOWSHIP-ROMBERG SIGN	PREOPERATIVE DIAGNOSIS	OPERATIVE PROCEDURE	COMPLICATIONS AND ASSOCIATED DISEASE	SIDE OF HERNIA	RESULT
1 1940	75 C. F	Sudden epigastric pain, nausea, vomiting, cramping; emaciated, wt. loss 10-15 lbs. previous 12 months; no masses; tender midabdomen, tympanitic, distended	Not recorded	Intestinal obstruction, GI malignancy, mesenteric vascular occlusion	Local anesthesia; rt. paramedian incision; 1 cm. left obturator defect, loop of gangrenous ileum; resection with side-to-side anastomosis; defect not closed	Arteriosclerotic heart disease, severe	Left	Died
2 1947	37 C. F	Pain RLQ—7 hours, vomiting—2 hours, pain down rt. leg; operation 3 previous times, one for intestinal obstruction 3 months earlier; recurrent attacks of colic and mild weight loss since	Not recognized	Recurrent small bowel obstruction	Operation delayed by suction treatment, 1½ days; general anesthesia; rt. lower rectus incision, tightly incarcerated loop of ileum in 1½ cm. defect, spill from 3 cm. area of gangrene; resection and end-to-end anastomosis; defect closed with 3 mattress sutures No. 2-0 silk	Wound infection	Right	Lived
3 1947	81 C. F	Abdominal pain and pain down rt. leg, recurrent cramping pain, nausea, vomiting; thigh pain aggravated by movement for 1 year; 3 previous attacks; emaciated	+	Obstructed obturator hernia	Local anesthesia; low McBurney incision, complete obstruction with 6-inch loop of ileum, mesenteric defect 2.5 by 1 cm., had smaller left obturator hernia defect; both closed adequately with No. 3-0 silk	Chronic bronchitis, arteriosclerotic heart disease, congestive heart failure	Right (left defect also present)	Lived
4 1950	61 C. M	Asymptomatic; elective bilateral femoral hernia repair	-	Bilateral femoral hernia	Spinal anesthesia; Cheatle-Henry approach, left obturator hernia, sac ligated, fascia closed over opening with interrupted cotton, large rt. obturator defect; no sac	None Known diaphragmatic hernia	Left	Lived
5 1951	76 C. F	Obstipation 5 days, vomiting 4 days, no abdominal pain, emaciated, marked distention, succussion splash; nontender	+ Not recognized	Mechanical small bowel intestinal obstruction	General anesthesia; rt. rectus incision; strangulated rt. obturator hernia; defect less than 1 cm., Richters' hernia; Summers stitch to invert tiny area of gangrene; defect closed with interrupted cotton; left side normal	Wound infection, malnutrition, cerebral vascular accident	Right	Died
6	81	One week of cramping,	-	Mechanical small	Rt. mid-rectus, muscle split-	Senile psychosis	Left	Lived

					tiny area of gangrene; defect closed with interrupted cotton; left side normal			
6 1951	81 C. F	One week of cramping, vomiting, obstipation. No past history of obstruction, distended; succussion splash	–	Mechanical small bowel obstruction	Rt. mid-rectus, muscle splitting incision; general anesthesia; Richters' hernia terminal ileum—viable; repair with interrupted No. 24 cotton	Senile psychosis	Left	Lived
7 1951	85 C. F	Cramping abdominal pain, 4 days; one similar episode 1 year prior; distended, nontender abdomen; fecal vomiting 2 days	–	Probable strangulated femoral hernia	Right inguinal incision; local anesthesia; Richters' hernia in rt. obturator defect; viable ileum reduced; one stitch No. 24 cotton	Advanced generalized arteriosclerosis Bronchopneumonia	Right	Lived
8 1951	85 C. F	Constipation 2 weeks; cramping pain, nausea, vomiting 2 days; distended, hyper-tympanitic abdomen	–	Large bowel obstruction	Right rectus incision; local anesthesia; loop of terminal ileum	None	Right	Lived
9 1951	62 C. M	Elective femoral hernia repair; asymptomatic	–	Right femoral hernia	Cheatle-Henry approach; incidental rt. obturator hernia; herniated extraperitoneal fat only in hernia; closed with No. 24 cotton	None	Right	Lived
10 1952	68 C. F	Vomiting 2 days, pain denied; no masses, slight distention, succussion splash; no scars or hernia	–	Small bowel obstruction (?), internal hernia (?), mesenteric vascular occlusion	Local anesthesia, rt. rectus incision; gangrenous necrotic, perforated ileum 3 feet from ileocecal valve, fecal spill; resection with end-to-end anastomosis; closed defect with No. 24 cotton using lateral peritoneum and infundibulopelvic ligament	Peritonitis Parotitis	Right	Lived
11 1953	85 C. F	Nausea, vomiting, cramping 5 days; sudden onset pain down rt. leg, no past history of obstruction	+ Not recognized	Mechanical small bowel obstruction	Low rt. paramedian incision; gangrenous, black loop of ileum ruptured by taxis; resection and end-to-end anastomosis; defect closed with 4 interrupted No. 24 cotton mattress sutures	Wound infection; congestive heart failure, cerebral vascular accident	Right	Died
12 1958	75 C. F	Symptoms nausea, vomiting, cramping pain 7 days, fecal vomiting 2 days, lower abdominal distention	– Not ilicited	Mechanical small bowel obstruction, adhesive band	Rt. rectus incision, general anesthesia; dense adhesions, rt. obturator hernia with Richter's incarceration of wall of ileum in 2 cm. defect, not gangrenous; repair with No. 24 cotton	None	Right	Lived

examined if the patient is in suitable condition. Cases 3 and 4 in this series had bilateral obturator defects. Coexistent femoral defects will also be found in a certain number of cases (Cases 4 and 9).

In the majority of earlier cases no attempt was made to repair the fascial defect remaining after release of the sac and its contents. Throckmorton[29] has stated that no attempt has been made to close the defect because it "was not amenable to closure by suturing." Experience at the Los Angeles County General Hospital has shown that in virtually every case the fascial hernia defect has been closed by one or more interrupted silk or cotton sutures (Table I and Fig. 1, *a* and *b*). These patients are usually extremely ill and lengthy repairs are both unnecessary and unwise.

A great variety of techniques have been used for closure of the obturator canal defect. Many of these are summarized in Table II. Since simply narrowing the entrance of the foramen with interrupted sutures after reduction of the sac and contents works so well, it is surprising to find so many different methods described, many of which are relatively difficult and time-consuming. Harper and Holt[14] have stated that a simple purse-string suture of the neck of

TABLE II. SUMMARY OF METHODS USED FOR CLOSURE OF OBTURATOR HERNIA DEFECTS

Short[25]	1923	Plug of rib cartilage
Horine[17]	1927	Suture sac to peritoneum after inverting apex of sac through defect
Grey-Turner[13]	1938	Evert sac and ligate base
Franklin[10]	1938	Plug of rib cartilage
Wakeley[32]	1939	Ligate sac, then suture strip from innermost fibers of pectineus muscle to periosteum of obturator canal
Throckmorton[29]	1950	Patch of tantalum gauze
Pender[19]	1950	Plug of tantalum gauze placed through thigh incision
Rothman[22]	1951	Patch of peritoneum, 1 by 1½ inches
Stone and McLanahan[28]	1954	Osteoperiosteal flap turned back from inner pubic bone over opening
Harper and Holt[14]	1956	Closed peritoneum over opening from below upward, incorporating base of sac
Gilfillan[12]	1958	Everted and ligated sac; filled defect with plug of free omentum
Rogers (author)	1959	Patch of Teflon cloth

the sac will usually be adequate. They have closed the peritoneum over the opening, incorporating the base of the sac in the sutures. This is undoubtedly sufficient since most of the defects are small, but it differs from most hernia repairs in which reduction and resection of the sac is a fundamental part of the repair. Other surgeons have plugged the canal with cartilage[25] or with free omentum.[12] An osteoperiosteal flap was advocated by Stone and McLanahan.[28] Others have everted the sac and ligated the base to complete a repair.[13] Throckmorton[29] and Pender[19] have each reported separately on the use of tantalum gauze in the repair. Pender used such gauze to plug the opening through a thigh incision, while Throckmorton patched the opening from above. In 1951 Rothman[22] described a repair using a patch of peritoneum.

Some large defects will occasionally be found which will resist closure by simple suture. The author has found a patch of Teflon or Dacron cloth an

excellent and readily available material to cover such larger defects. Fig. 2, *a* and *b* illustrate the use of this method. It is an ideal material to work with since it sews without fraying, is easily cut to any pattern desired, and is long lasting without causing excessive scarring. Other similar prosthetic materials would be as satisfactory.

Results of Treatment.—Mortality rates for treatment of acute obturator hernias have been high. The hernia so frequently occurs in aged and debilitated people in whom diagnosis is made improperly and late in the course of their illness. Many of the patients in this series had serious associated diseases which further added to their risk of treatment. There is a high incidence of gangrenous bowel. The fatality rate earlier has been above 50 per cent in many collected series. Horine[17] noted 27 deaths in 47 cases in 1927 and Watson[34] reported on a group of 149 cases in 1939 with a 48 per cent mortality figure.

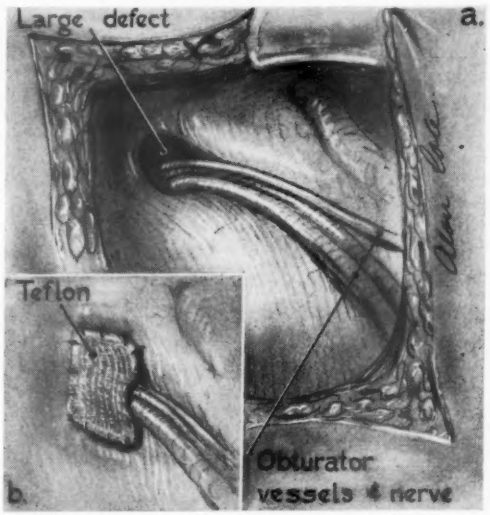


Fig. 2.—*A*, Unusually large obturator defects may present a problem in closure. *B*, A simple method of repair is illustrated with use of a patch of Teflon.

All the patients in the present series were operated upon with only 3 deaths —a 30 per cent mortality for cases of acute strangulation. There was gangrenous small bowel in 5 of the 10 acute cases reported here (Cases 1, 2, 5, 10, 11). Four of these required segmental resections, whereas in the fifth case the gangrenous area was inverted with a Summer's stitch. Three of these patients succumbed and were the only deaths in the series. Six of this group had severe associated disease involving pulmonary or cardiovascular systems which, along with their advanced age, added heavily to their risk of treatment. Local anesthesia was employed in 5 patients, indicating their unusually poor condition. Three developed wound infections.

SUMMARY

A group of 12 cases of obturator hernia is presented, in 10 of which there was acute incarceration. The history, mechanism, and symptomatology of obturator hernia is reviewed and illustrated. The majority of patients who develop obturator hernias are elderly, weak, and often emaciated. The defect is

more common in women than men (6:1). The average age of the patients in the present series was over 70 years. There were 10 females and 2 males, the youngest being a 37-year-old woman.

The chief problem in medical care has been the failure to consider strangulated obturator hernia and to recognize the significance of pain or disturbance along the path of the obturator nerve (Howship-Romberg sign). Although not always present, this sign can be pathognomonic in a patient otherwise exhibiting the classical signs of small bowel obstruction.

Early surgery with release of the sac and simple closure of the defect represents ideal treatment. If a diagnosis can be correctly made preoperatively, a suprapubic, retroperitoneal (Cheatle-Henry) approach through a vertical or transverse incision is recommended. The majority of obturator hernias contain terminal ileum and are right-sided (2:1). There is a high incidence of incarcerated nonviable bowel and the Richter's type of hernia is common. Defects are bilateral in about 6 per cent of the cases.

Where the obturator defect is large and simple fascial approximation not adequate for closure, a patch of prosthetic cloth (Teflon, Dacron) can be simply and effectively applied.

Gangrenous bowel was encountered in 5 cases in this series. There were 3 deaths. In 2 cases, bilateral defects were present.

Strangulated obturator hernia is a rare but particularly lethal obstruction because of its occurrence in the elderly, the high incidence of gangrenous intestine, and low incidence of correct diagnosis.

REFERENCES

1. Adams, H. D., and Smith, D. C.: Obturator Hernia, J. A. M. A. **137**: 948-950, 1948.
2. Anson, B. J., McCormack, L. L., and Cleveland, H. C.: Anatomy of Hernial Regions: Obturator Hernia and General Considerations, Surg. Gynec. & Obst. **90**: 31-38, 1950.
3. Arnaud, G.: Dissertation on Hernias, translated by A. Miller, London, 1748.
4. Arnaud, P. R.: *In* de Garengeot, R. J. C.,[11] p. 709
5. Cheatle, G. L.: An Operation for the Radical Cure of Inguinal and Femoral Hernia, Brit. M. J. **2**: 68, 1920.
6. Desmond, A. M., and Hutter, F.: Strangulated Obturator Hernia, Brit. J. Surg. **35**: 318-320, 1948.
7. Duverney, M.: *In* de Garengeot, R. J. C.,[11] p. 709.
8. Erichsen, J. E.: Select Cases of Hernia, Lancet **1**: 10-12, 1850.
9. Erichsen, J. E.: Science and Art of Surgery, ed. 8, Philadelphia, 1885, Lea Bros., vol. 2, p. 803.
10. Franklin, R. H.: Obturator Hernia, Lancet **1**: 721-722, 1938.
11. de Garengeot, R. J. C.: Memoire sur plusiers hernies singulieres, Mem. Acad. roy. de chir., Paris **1**: 699-716, 1743.
12. Gilfillan, R. R.: Obturator Hernia, Canad. J. Surg. **1**: 366-367, 1958.
13. Grey-Turner: Quoted by R. H. Franklin.[10]
14. Harper, J. R., and Holt, J. H.: Obturator Hernia, Am. J. Surg. **92**: 562-565, 1956.
15. Henry, A. K.: Operation for Femoral Hernia by a Midline, Extraperitoneal Approach, Lancet **1**: 531, 1936.
16. Heu-Hsiang: Strangulated Obturator Hernia: Report of a Case, Chinese M. J. **75**: 328, 1957.
17. Horine, C. F.: Obturator Hernia, Ann. Surg. **86**: 776-781, 1927.
18. Obre, H.: Operation des hernies, Med. chir. Tr. London **34**: 233-238, 1851.
19. Pender, B. W. T.: Recurrent Obstruction of Obturator Hernia, Brit. M. J. **2**: 1038, 1950.
20. Penworth, P.: Obturator Hernia, Am. J. Surg. **71**: 539-541, 1948.
21. Rose, T. F.: Obturator Hernia, M. J. Australia **1**: 13, 1954.

22. Rothman, M.: The Repair of Obturator Hernia With Peritoneal Graft, New York J. Med. **51**: 1186, 1951.
23. Rowe, E. B., and Bynum, G. A.: Obturator Hernia, Am. Surgeon **18**: 770-777, 1952.
24. Sabeeley, E.: Obturator Hernia, Am. J. Surg. **65**: 422-424, 1944.
25. Short, A. R.: The Treatment of Strangulated Obturator Hernia, Brit. M. J. **1**: 718, 1923.
26. Singer, R., Leary, P., and Hofmeyer, N. G.: Obturator Hernia, South African M. J. **29**: 73-75, 1955.
27. Soothill, E. F.: Obturator Hernia, Guy's Hosp. Rep. **103**: 43, 1954.
28. Stone, H., and McLanahan, S.: Obturator Hernia *in* Lewis' Practice of Surgery, Hagerstown, Md., 1954, W. F. Prior Co., vol. 7, chapt. 9, pp. 68-69.
29. Throckmorton, T. D.: Repair of Obturator Hernia With a Tantalum Gauze Implant, SURGERY **27**: 888-892, 1950.
30. Van Meurs, D. P.: Strangulation of the Ovary and Fallopian Tube in an Obturator Hernia, Brit. J. Surg. **32**: 539, 1945.
31. Van Zwalenburg, C.: Final Report on Case of Radical Operation for Cure of Double Obturator Hernia, Surg. Gynec. & Obst. **33**: 428-430, 1921.
32. Wakeley, C. P. G.: Obturator Hernia, Brit. J. Surg. **26**: 515, 1939.
33. Waterfall, M. C.: Recovery From Strangulated Obturator Hernia, Brit. M. J. **1**: 585, 1950.
34. Watson, L. F.: Hernia, ed. 3, St. Louis, 1939, 1948, The C. V. Mosby Company.

THE ROLE OF CEREBRAL EDEMA IN ISCHEMIC CEREBRAL NEUROPATHY AFTER CARDIAC ARREST IN DOGS AND MONKEYS AND ITS TREATMENT WITH HYPERTONIC UREA

HARRY A. KAUPP, JR., M.D., ROBERT E. LAZARUS, B.S., NICHOLAS WETZEL, M.D., PH.D., AND THOMAS E. STARZL, M.D., PH.D.,* CHICAGO, ILL.

(From the Department of Surgery, Northwestern University Medical School)

THE USE of hypothermia to promote neurologic recovery after cardiac arrest has been based on the belief that the efficacy of this therapy is due to the consequent reduction in cerebral edema.[8, 10] If this were true, hypertonic urea, which has been shown by Javid[1, 2] to be a remarkably effective agent in the treatment of cerebral edema, would be expected to have a similarly beneficial effect upon recovery. The present study is concerned with the experimental evaluation of this drug in the treatment of simulated cardiac arrest in dogs and monkeys.

During the experimentations, considerable data were accumulated on the maximum tolerance to cardiac arrest of the normothermic dog and monkey brain under optimum prearrest and resuscitative circumstances. This material has been analyzed in some detail since the period of safe total cerebral ischemia in both species has been longer than previously reported.

METHODS

Experiments were performed on 60 mongrel dogs and 19 *Macaca mulatta* monkeys. The animals were anesthetized with 25 to 30 mg. per kilogram of sodium pentobarbital and placed on a respirator[4] supplied with 100 per cent oxygen. The animal temperatures were maintained at 37° C. with heating blankets during and after operation. Except for maintenance of body temperature and treatment with urea† or placebo solution, no postoperative care was provided after the animals were removed from the respirator. Other specific details of methodology are presented under the appropriate sections.

OPERATIVE TECHNIQUE FOR SIMULATED CARDIAC ARREST

The operative procedure is essentially the same for dogs and monkeys (Fig. 1). A thoracotomy is performed through the left fourth intercostal space. The aortic arch and its 2 branches (brachiocephalic trunk and left subclavian artery) are dissected free. Tapes are passed about the aorta, above the level of the first intercostal artery, and around the 2 branches of the arch. The superior and inferior venae cavae are then encircled with tapes, the former above the entrance of the azygos vein. The venae cavae, aorta, and the 2 arch branches are then occluded simultaneously (Fig. 1). When occlusion is terminated, the clamps are released simultaneously and the chest closed immediately with water-seal drainage.

During the period of cerebral ischemia, a diminutive systemic and pulmonary circulation is maintained. The only venous inflow to this system is from the azygos vein and the only

Aided by a grant from the American Heart Association and a grant from the Chicago Heart Association.

Received for publication Jan. 20, 1960.

*Markle scholar.

†The urea used in this study was provided in crystalline form by the Abbott Laboratories, North Chicago, Ill.

systemic outflow is to the coronary arteries. In some cases the inflow appeared to be too great and vascular engorgement of the lungs slowly developed but, after release of the clamps, this disappeared immediately. The operative mortality rate in dogs was 5 per cent (due to ventricular fibrillation) and the procedure was especially well tolerated in monkeys in which there were no immediate deaths in 19 experiments. In 2 monkeys the occlusion was maintained for 60 and 90 minutes with maintenance of a strong heartbeat and restoration of normal circulation at the termination of occlusion.

Because the time of ischemia necessary to produce neurologic injury was longer than previously reported, a special effort was made to preclude the possibility that arterial blood was reaching the brain in some circuitous manner. In 2 monkeys, methylene blue was injected into the left ventricle. No dye reached either the spinal cord or brain. In 3 monkeys and several dogs, the brain was removed during occlusion so that the vertebral and cerebral arteries as well as the circle of Willis could be transected under direct vision. No arterial inflow whatever was present.

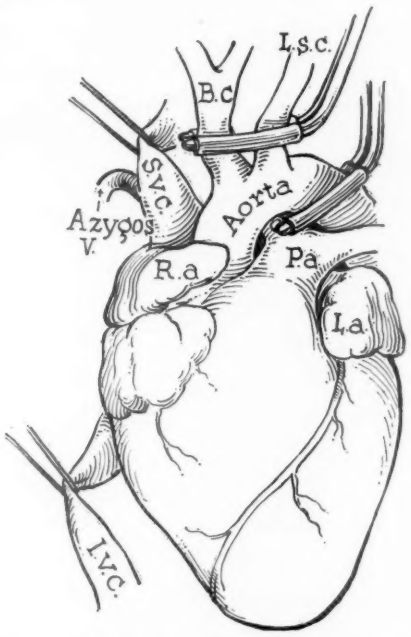


Fig. 1.—Method of simulated cardiac arrest. Operation is performed through the left chest. Note that circulation is retained to the coronary arteries.

RESULTS

Factors That Influence the Period of Safe Occlusion in Dogs.—Certain obvious factors will materially increase the period of occlusion necessary to cause permanent central nervous system damage. These include avoidance of partial occlusion of the venae cavae prior to complete test occlusion, insurance of a vigorous heartbeat at the conclusion of ischemia, and the use of 100 per cent oxygen. It was found, for example, that the time of ischemia necessary to produce brain injury was about 4 minutes less when the animal was respirated with compressed air than when oxygen was used.

More subtle influences were also important. At the beginning of the work a commercial respirator was used, supplied with 100 per cent oxygen. The necessary occlusion for brain injury in 10 dogs was 10½ minutes, a time comparable to that reported by Marshall[5] and Zimmerman[10] and their coworkers. Ultimately the electronic respirator designed by Lewis[4] was employed,

with the result that the time of ischemia for comparable injury was increased to 14 minutes. It was also found that efforts to promote early spontaneous respiration after the chest was closed led to a sharp rise in the mortality rate and an unpredictable reduction in the time necessary for occlusion. Consequently, the animals were maintained on the respirator for as long as 2 to 8 hours postoperatively and were removed only when they began to fight the mechanical control of their respiration.

Time of Complete Cerebral Ischemia in Dogs With Complete Recovery.—Under the optimum conditions outlined above, 6 dogs were subjected to complete cerebral ischemia for 10 minutes. All recovered (Table I). Four dogs had occlusions for 11 minutes and 4 for 12 minutes. All of these animals eventually recovered but about one half required 24 to 48 hours before normal locomotor control was restored. Seven dogs were subjected to a period of occlusion for 13 minutes. Three of these did not recover completely, ataxia and drowsiness persisting for at least 1 week after operation. With occlusion for 14 minutes, two thirds of the animals either died from profound central nervous system injury (opisthotonus, extensor rigidity, coma) or manifested permanent neurologic deficit such as sleepfulness, ataxia, or head nodding (Table I).

TABLE I. THE RESULT OF DIFFERENT PERIODS OF COMPLETE CEREBRAL ISCHEMIA IN DOGS

MINUTES OF OCCLUSION	NO. OF ANIMALS	RECOVERED	DIED OR PERMANENT INJURY
10	6	6	0
11	4	4	0
12	4	4	0
13	7	4	3
14	18	4	14

Effect of Urea on Recovery After Cerebral Ischemia in Dogs.—Thirty-seven dogs were subjected to 14 minutes of complete cerebral ischemia. Nineteen animals were given 30 per cent urea intravenously, made up in 5 per cent glucose/normal saline. The infusions were given 30 minutes after operation and every 12 hours thereafter until recovery was complete or until 3 days had passed. The dose was 1 Gm. of urea per kilogram and the infusions were given over a 30-minute period. Eighteen dogs were used for controls. Treatment was exactly the same in these animals except that 5 per cent glucose/normal saline infusions without urea were used.

The results with and without treatment with urea are shown in Table II. Six of the 19 dogs treated with urea recovered completely within 1 week. Three others recovered sufficiently to eat and survive indefinitely, but manifested ataxia and other findings of extrapyramidal injury. Ten of the 19 dogs died with profound neurologic injury.

In the untreated group, 4 of 18 controls recovered completely, 2 had incomplete recovery, and 12 died. Analysis of the results did not demonstrate statistically significant protection of the treated as compared to the untreated animals.

TABLE II. EFFECT OF TREATMENT WITH UREA ON RECOVERY AFTER 14 MINUTES OF CEREBRAL ISCHEMIA IN DOGS

	TREATMENT WITH UREA	TREATMENT WITH PLACEBO
No. of animals	19	18
Complete recovery	6	4
Permanent injury	3	2
Died	10	12

Time of Complete Cerebral Ischemia in Monkeys.—Single monkeys were subjected to occlusion for 8, 10, and 12 minutes. Although recovery from anesthesia was slow, all animals were neurologically normal within 24 hours. Seven monkeys had occlusions for 14 minutes (Table III) with an immediate mortality rate of more than 50 per cent.

TABLE III. THE RESULT OF DIFFERENT PERIODS OF COMPLETE CEREBRAL ISCHEMIA IN MONKEYS

MINUTES OF OCCLUSION	NO. OF ANIMALS	RECOVERED	DIED OR PERMANENT INJURY
8	1	1	0
10	1	1	0
12	1	1	0
14	7	2	5

The animals with 14 minutes of cerebral ischemia responded very sharply to this insult, either with complete recovery or with early death. In contrast to the dogs, in which a significant number of the animals lived despite serious neurologic deficit, none of the monkeys which did not recover normal locomotor control lived beyond 24 hours. These animals were obtunded or comatose, with sluggish or absent response to pain. Opisthotonus and extensor rigidity were occasionally seen but, generally, the stricken monkey was areflexic, with absent grasp and righting reflexes. In some cases, the monkeys could never be removed from the respirator, and in others they would assume an immobile position for many hours with increasing weakness and death.

Effect of Urea on Recovery After Cerebral Ischemia in Monkeys.—Fourteen minutes of cerebral ischemia was imposed on 14 monkeys. Seven animals were treated with 30 per cent urea (1 Gm. per kilogram), and the other 7 with placebo injections. The results are shown in Table IV.

TABLE IV. EFFECT OF TREATMENT WITH UREA ON RECOVERY AFTER 14 MINUTES OF CEREBRAL ISCHEMIA IN MONKEYS

	TREATMENT WITH UREA	TREATMENT WITH PLACEBO
No. of animals	7	7
Complete recovery	3	2
Permanent injury	0	1
Died	4	4

Four of the 7 controls died in from 7 to 24 hours after operation. Two of the controls recovered completely with no demonstrable harmful effects. One monkey apparently recovered completely, but a remarkable behavioral change soon became manifest. The animal had had a savage personality

before operation, but postoperatively he was docile and affectionate. He soon developed an appetite for meat, a characteristic finding of bilateral temporal lobe destruction (Küver-Bucy syndrome[3]). At the autopsy 16 weeks after operation, no gross lesion of the temporal lobes was found.

Seven monkeys were treated with urea. Four animals died in from 8 to 16 hours. The other 3 recovered completely. Convalescence in the animals that recovered was characterized by a steady improvement in which there was no detectable change immediately after each urea injection. Similarly, urea produced no alteration in the state of wakefulness or responsiveness in the monkeys that died.

Autopsies were performed on all monkeys. The control animals all had cerebral edema with swollen gyri and tight approximation of the cortex to the dura. In the animals treated with urea the brains were slack and generally only remarkable for their lack of swelling.

DISCUSSION

Numerous methods have been used for experimental study of cerebral ischemia. Techniques which only involve occlusion of the vertebral and carotid arteries, either in the neck or the chest, provide inconstant results since an unknown quantity of blood reaches the brain through intercostal and other collateral pathways. The method used in the present study is a modification of the inflow-outflow occlusion method of Marshall and associates[5] in which coronary arterial circulation is maintained during the time when all other systemic outflow is completely blocked. No arterial blood whatever reaches the brain during occlusion, and the test periods are sharply defined since the condition of the heart remains excellent before, during, and after occlusion. In our experience with occlusion through a right thoracotomy, as described by Marshall,[5] the incidence of cardiac deterioration during the test period was high presumably because the aortic clamp must be applied too close to the coronary arteries. With inflow-outflow occlusion from a left approach, as employed in the present study, this complication was virtually eliminated.

In human beings the duration of cardiac arrest necessary to produce permanent neurologic injury is not known since the exact moment of arrest is difficult to determine, the catastrophe is usually preceded by variable periods of anoxia, shock, or other physiologic derangements, and the circulatory state is usually not ideal for some time after resuscitation has been completed.[7] Many authors have stated that 3 to 5 minutes constitute the time limits beyond which cardiac arrest can be expected to cause neurologic sequellae. In the present study, it has been shown that complete cerebral ischemia can be tolerated in almost every case for 12 minutes by both the dog and monkey under the appropriate circumstances. These conditions include the presence of a normal cardiovascular state before and after ischemia, the use of oxygen, employment of an efficient respirator, and prolonged mechanical support of respiration postoperatively. The elimination of any one of these factors markedly reduces the period of cardiac arrest to which the brain can be safely subjected.

Recently, hypothermia has been proposed[8, 10] as adjunctive postoperative treatment for patients who have been resuscitated from cardiac arrest in an effort to forestall the neurologic sequellae which are so often a late cause of death. The efficacy of this therapy has been assumed to be due to a reduction in brain volume at the lowered temperature.[8, 10] Hypertonic urea has been shown by Javid[2, 9] to be an effective form of therapy for cerebral edema. Indeed, in the present study, animals treated with this agent did not have autopsy evidence of brain swelling and those not treated had clearly demonstrable evidence of cerebral edema. In spite of this successful treatment of cerebral edema, there was no material reduction in the mortality rate or neurologic disability in the treated animal. From this it is concluded that cerebral edema is an over-emphasized factor in the post-cardiac arrest neurologic syndrome, and that any benefits accruing from the use of hypothermia are due largely to other factors. Strong collateral evidence favoring this conclusion is provided by a recent work of Rosomoff and his co-workers.[6] These authors demonstrated that hypothermia, if applied for a brief period within a few hours after acute experimental brain injury, resulted in a striking reduction in mortality. Evidence was presented[6] that the efficacy of this treatment was not due to reduction of cerebral edema (and cerebrospinal fluid pressure) since both the treated and untreated animals had comparable cerebral edema.

SUMMARY

1. The value of hypertonic urea in forestalling the neurologic sequellae after cardiac arrest has been assessed in dogs and monkeys. Simulated cardiac arrest was produced with an inflow-outflow occlusion technique in which the blood supply to the heart was maintained during the period of complete cerebral ischemia.

2. A number of factors increase the duration of cardiac arrest which can be tolerated without gross neurologic sequellae. These include the presence of an effective circulatory state before and after the arrest, the administration of 100 per cent oxygen before and after the test period, the use of an efficient respirator, and avoidance of premature efforts to promote spontaneous respirations postoperatively.

3. Under optimum conditions almost all dogs and monkeys will recover completely after 12 minutes of simulated cardiac arrest. With longer occlusions, death and serious neurologic morbidity occur.

4. Monkeys and dogs were subjected to simulated cardiac arrest for 14 minutes. Hypertonic urea did not materially decrease either the mortality rate or neurologic morbidity in either species, as compared to the recovery rate in controls, despite the fact that the urea-treated animals had demonstrably less brain swelling.

5. From this it is concluded that cerebral edema is an overemphasized factor in the post-cardiac arrest syndrome, and that the principal deterrent to recovery is anoxic injury to the neurons which is not beneficially influenced by reduction of brain volume.

REFERENCES

1. Javid, M.: Urea: New Use of an Old Agent, S. Clin. North America 38: 907, 1958.
2. Javid M., Settlage, P., and Monfore, T.: Urea in the Management of Increased Intracranial Pressure, S. Forum 7: 528, 1957.
3. Küver, H., and Bucy, P. C.: Preliminary Analysis of Functions of the Temporal Lobes in Monkeys, Arch. Neurol. & Psychiat. 42: 979, 1939.
4. Lewis, F. J.: An Electronically Controlled Respirator for the Experimental Laboratory, J Thoracic & Cardiovasc. Surg. 38: 408, 1959.
5. Marshall, S. B., Owens, J. C., and Swan, H.: Temporary Circulatory Occlusion to the Brain of the Hypothermic Dog, A. M. A. Arch. Surg. 72: 98, 1956.
6. Rosomoff, H. L., Shulman, K., Raynor, R., and Grainger, W.: Experimental Brain Injury and Delayed Hypothermia, Surg. Gynec. & Obst. 110: 27, 1960.
7. Stephenson, H. E.: Cardiac Arrest and Resuscitation, St. Louis, 1958, The C. V. Mosby Company.
8. Williams, G. R., and Spencer, F. C.: The Clinical Use of Hypothermia Following Cardiac Arrest, Ann. Surg. 148: 462, 1958.
9. Young, W. P., and Javid, M. J.: Study of the Use of Intravenous Urea After Simulated Cardiac Arrest in Dogs, S. Forum 10: 522, 1959.
10. Zimmerman, J. M., and Spencer, F. C.: The Influence of Hypothermia on Cerebral Injury Resulting From Circulatory Occlusion, S. Forum 9: 216, 1958.

VARIATIONS OF THE FLEXOR DIGITORUM SUPERFICIALIS (SUBLIMIS)

A. Travill, M.B., B.S., M.R.C.S., L.R.C.P.,* and E. Sandborn, M.D.,**
Kingston, Ontario

(From the Department of Anatomy, Queen's University)

BOTH anatomists and surgeons are agreed that the flexor digitorum superficialis muscle of the forearm is subject to considerable variation, although major anomalies are believed to be rare (Hollinshead[3]). Wood Jones[6] goes so far as to comment that "hardly any two subjects have this muscle in exactly similar form."

We undertook a consideration of this muscle and its variations upon finding 2 markedly distinct and unusual variations of it in 2 cadavers in a small group of dissecting room subjects.

The first subject had no tendon of the flexor digitorum superficialis to the fifth digit of both hands (Fig. 1, *A*). The tendon was replaced by a small muscle arising from the deep surface of the distal part of the flexor retinaculum. From this muscle a tendon passed distally to be inserted into the second phalanx of the little finger as would a normal tendon of the flexor digitorum superficialis.

The second subject showed the part of the flexor digitorum superficialis to the fifth digit arising as 2 distinct muscle bellies from either side of the distal part of the muscle (Fig. 1, *B*). Thence the very thin tendons from the muscle bellies passed into the palm where they joined together to form 1 tendon which continued onward to a normal division and insertion.

DISCUSSION

In the lower vertebrates the flexor digitorum superficialis is represented by 2 groups of muscles: a first group passing from the forearm to the palmar region, and a second group, the brevis muscles, passing from the palm to the fingers (Huber[4]). In the lemurs, the flexor digitorum superficialis is fused with the flexor digitorum profundus and the flexor digitorum brevis is still present (Bunnell[1]). All the accepted authorities in anatomy[1, 3-9] agree on the variability of the flexor digitorum superficialis but none quotes the percentage incidence of the variations. Furthermore, so far as we are aware, there has been no published report of the 2 variations just described, although Gray[2] described somewhat similar specimens. The usual variation commented upon is a complete absence of the fifth digital component of the flexor digitorum

Received for publication Jan. 22, 1960.
*Rehabilitation Foundation Fellow in Anatomy, Queen's University.
**Lecturer in Anatomy, Queen's University.

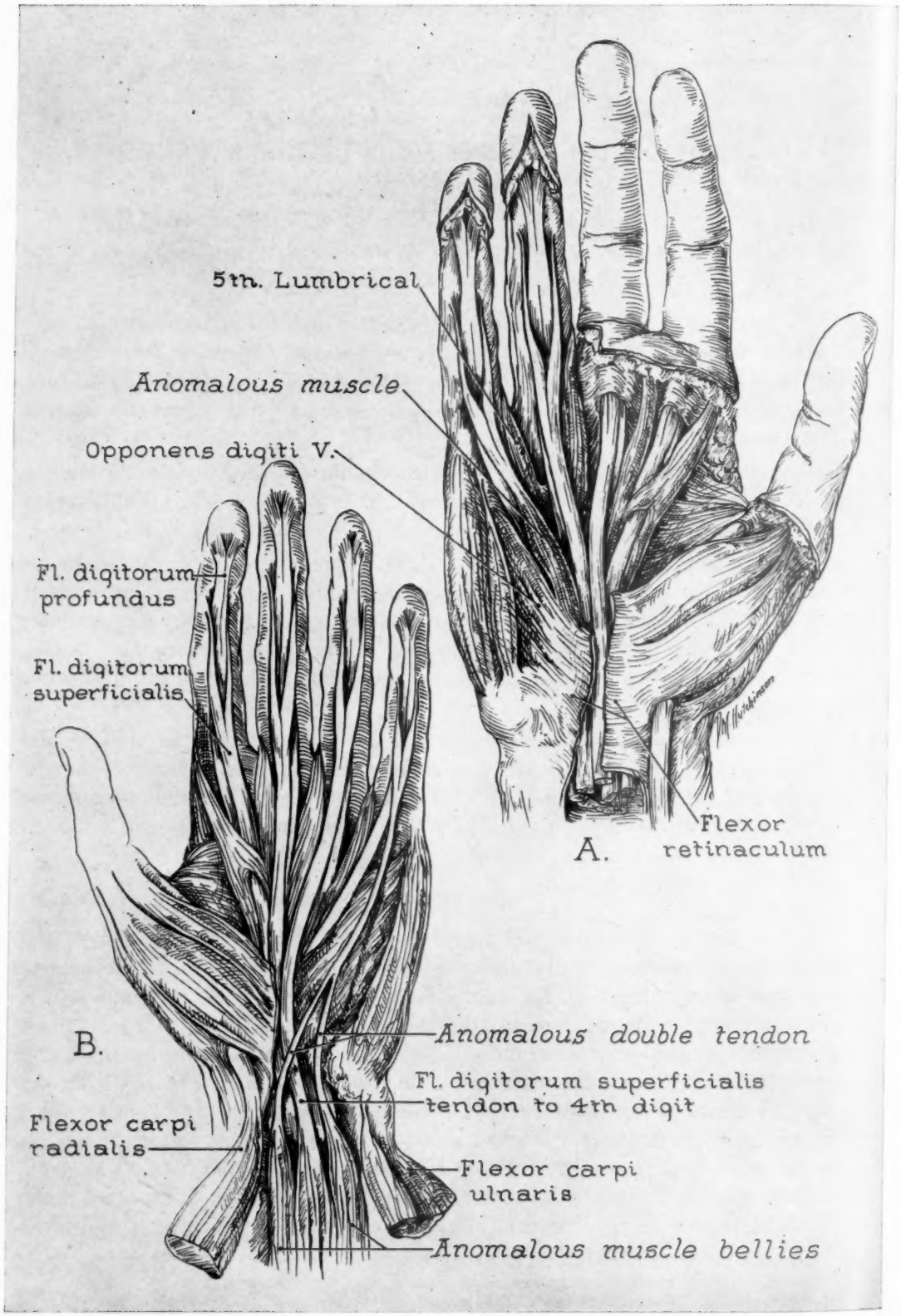


Fig. 1.—Two anomalies of the flexor digitorum superficialis.

superficialis. Other common variations seen are split tendons, fused tendons, absent tendons and, finally, fusion of the fleshy part of the flexor digitorum superficialis with that of the flexor digitorum profundus.

To the surgeon the flexor digitorum superficialis is a useful as well as an interesting muscle for 2 reasons. First, the separate tendons may be used individually for tendon transfers, grafts, and repairs; and, second, their occasional absence or loss does not materially impair the precision of the movements of the hand. Therefore the occurrence of variations of the flexor muscles as marked as those just noted indicates the necessity of alternative preoperative planning for surgery in this region.

REFERENCES

1. Bunnell, S.: Surgery of the Hand, ed. 3, Philadelphia, 1956, J. B. Lippincott Company.
2. Gray, D. J.: Some Variations Appearing in the Dissecting Room, Stanford M. Bull. 3: 120, 1945.
3. Hollinshead, W. H.: Anatomy for Surgeons, New York, 1958, Paul B. Hoeber, Inc., vol. 3.
4. Huber, G. C.: Piersol's Human Anatomy, Philadelphia, 1936, J. B. Lippincott Company, vol. 1.
5. Johnston, T. B., and Whillis, J.: Gray's Anatomy, ed. 31, London, New York and Toronto, 1954, Longmans, Green & Co., Inc.
6. Jones, F. Wood: Principles of Anatomy as Seen in the Hand, ed. 2, London, 1949, Baillière, Tindall & Cox.
7. Kaplan, E. B.: Functional and Surgical Anatomy of the Hand, Philadelphia, 1953, J. B. Lippincott Company.
8. Testut, L., and Laterjet, A.: Traité D'anatomie humaine, ed. 9, Paris, 1948, Gaston Doin Cie, vol. 1.
9. Thane, G. D.: Quain's Elements of Anatomy, ed. 10, London and New York, 1892, Longmans, Green & Co., vol. 2, part 2.

A SIMPLE STERNAL LIGATURE CARRIER

FRANCIS S. GERBASI, M.D., DETROIT, MICH.

(From Alexander Blain Hospital and Harper Hospital)

WITH the advent of open-heart surgery, there has come the necessity of splitting the sternum transversely or horizontally in order to obtain adequate exposure of the heart. One probably insignificant but annoying problem which I have encountered since I have been dividing the sternum has been its reapproximation. Breaking off needles in the sternum when attempting to pass

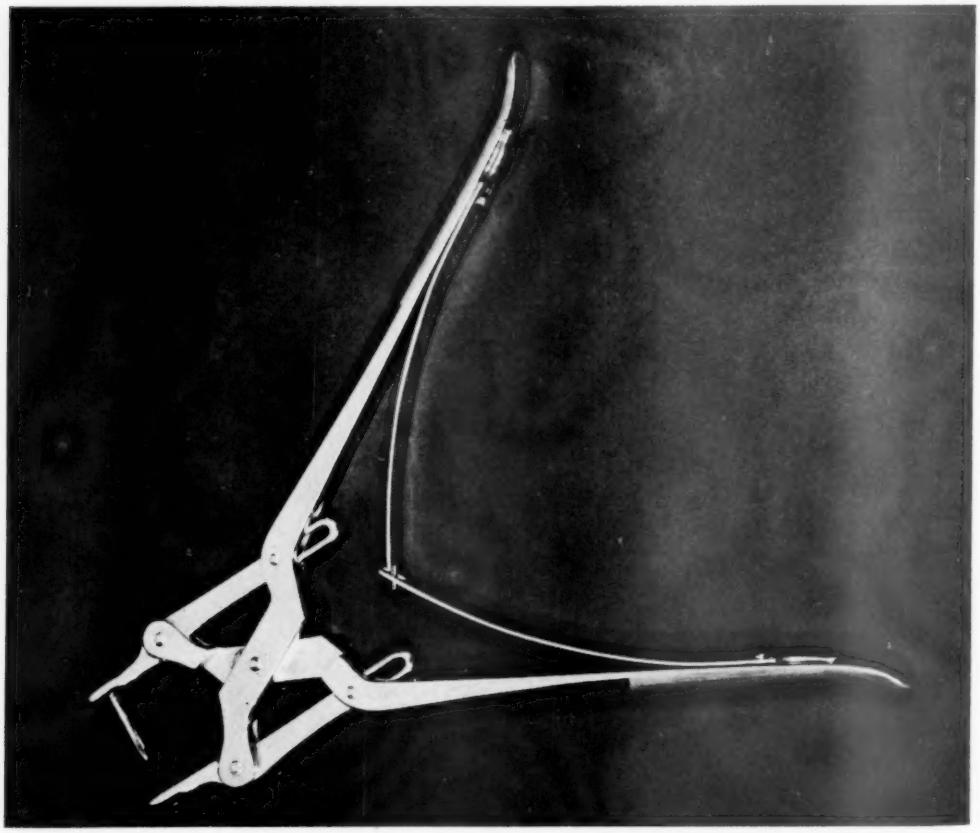


Fig. 1.

sutures through it has been a constant irritation. Drilling holes through this structure is time consuming, cumbersome, and usually necessitates making much larger holes than necessary. Therefore, a simple ligature carrier* was designed which would obviate this difficulty.

Received for publication Jan. 6, 1960.

*Manufactured by Ferndale Surgical Supply Company, 780 West Eight Mile Road, Ferndale, Mich.

DESCRIPTION

The ligature carrier is 28.5 cm. long when closed. It is made of stainless steel (Figs. 1 and 2). Two angled lever arms are pinned in a cross joint. Two punch arms are pinned to the ends of the lever arms. A sliding joint connects the distal end of the punch arm to a point on the lever arm distal to the crossed joint so that the 2 punch arms remain parallel at all times as the punch closes.

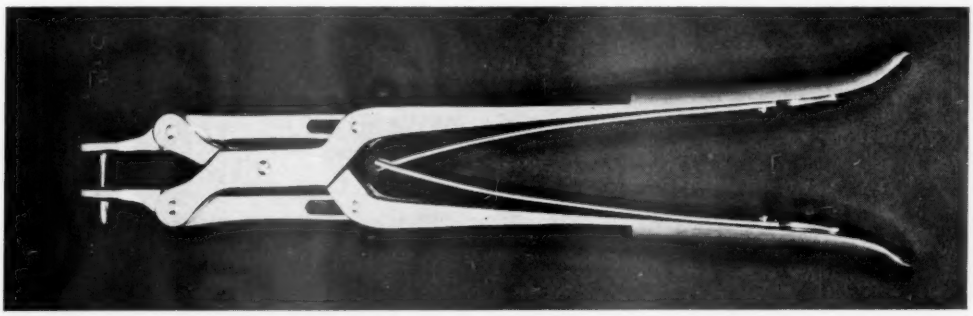


Fig. 2.

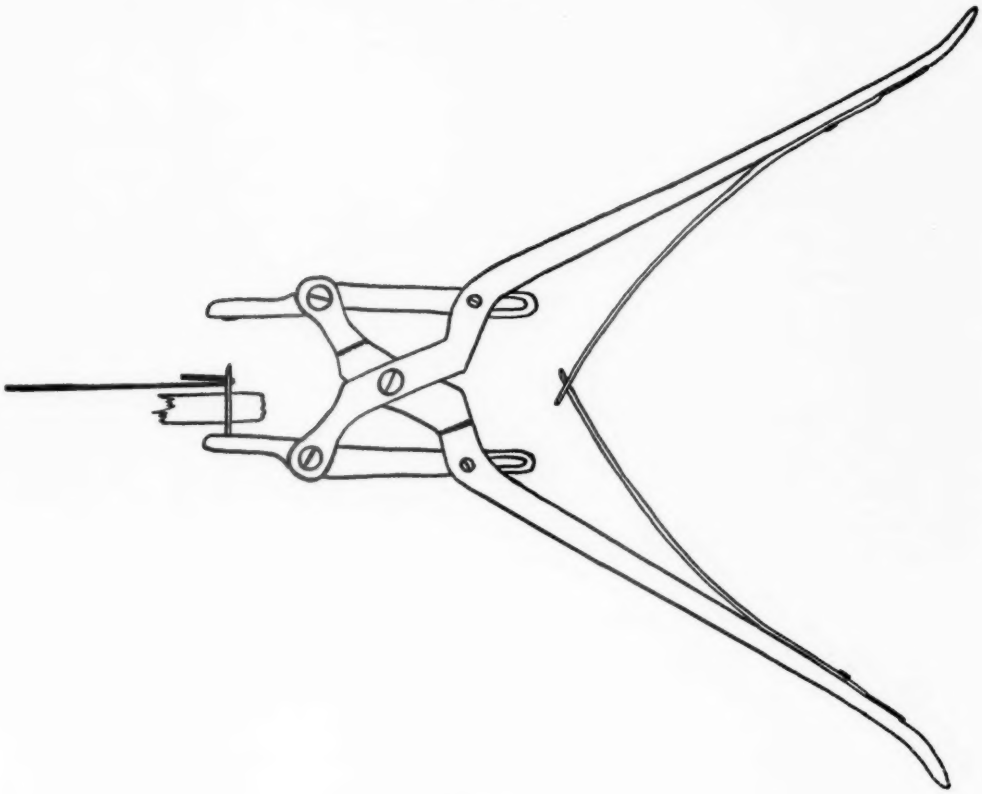


Fig. 3.

This is called "a parallel link mechanism." A sharp, perforated, triangular needle is inserted in the upper punch arm. There is a small opening in the head of the lower punch arm which allows the needle to pass through it. A double cantilever spring on the lever handles holds the punch open.

The lower punch arm is placed beneath the edge of the sternum. As the lever arms are approximated, the needle perforates the sternum, passing from the anterior surface posteriorily. The lower punch arm supports the posterior sternum. The needle passes through the opening in the head of the lower punch arm. The lever arms are released. The suture, which is usually wire, is passed through the eye of the needle. Only ½ in. (Fig. 3) is passed through the eye of the needle in order that this end of the suture will not remain in the sternum after the needle has been withdrawn. Thus, there is no difficulty in removing the suture from the needle.

The suture is not passed through the eye of the needle until after the clamp is released. This releases the lower arm of the clamp in order to protect the underlying heart from the point of the needle. The needle with its suture is withdrawn from the sternum, thus carrying the ligature through the sternum. The suture is removed from the needle. The opposite edge of the sternum is then perforated in the same manner. The clamp is released and the suture, which is on the under surface of the sternum, is threaded through the needle. The needle is then withdrawn from the sternum. The suture is now well placed, and can be tied or, if wire is used, can be twisted to hold the edges of the sternum together.

CONCLUSION

A simple ligature carrier has been described which facilitates the passage of ligatures through the sternum. It is hoped that it will eliminate the time-consuming, awkward, and sometimes frustrating moments one often encounters when he attempts to pass the conventional needle through the bony sternum.

CARELESS DRYING OF HANDS AS A POSSIBLE SOURCE OF CONTAMINATION: A PROPOSED SAFE TECHNIQUE

WILLIAM H. PRIOLEAU, M.D., CHARLESTON, S. C.

(From the Department of Surgery, Roper Hospital, and Medical College of South Carolina)

AFTER preparing the hands by a rigid scrubbing technique, surgeons not infrequently subject them to possible contamination by careless handling of the towel in the process of drying. When drying each arm from hand to elbow with a different end of a folded towel, the hanging end can easily become contaminated by contact with the scrub suit which, though freshly laundered, is not sterile (Fig. 1). The risk of contamination is increased if the shirt has been worn on the wards, at a preceding operation, or on the street in transit from residence or another hospital.

Fig. 1.

Fig. 2.

Fig. 1.—Incorrect. The hanging end of the towel is touching scrub shirt. Sleeves are long and wet.

Fig. 2.—Incorrect. Water from the elbow passes through 2 thicknesses of towel, as demonstrated by dye passing through towel to hand, and subjects hands to possible contamination.

Water on the elbow, coming in part from the upper arm above the level of thorough scrubbing, and at times from wet sleeves, presumably carries microorganisms and must be considered a source of contamination should it come in contact with the hand. That it passes readily through 2 thicknesses of towel can be demonstrated by coloring the water with dye (Fig. 2).

Received for publication Jan. 6, 1960.

In order to prevent contamination of the hands, the following method is proposed. The towel is carefully gathered into a ball so that it is under complete control. Both hands and the distal two-thirds of both forearms are dried (Fig. 3). A safe technique is to use the towel to dry only the hands and distal half of the forearms, permitting dripping and evaporation to take care of the elbow region.

When it is desired to use the towel for the elbow region (the usual practice according to our observation), the following method is proposed. The towel ball is grasped with the fingers of one hand and the opposite elbow is mopped. Both elbows are dried with the same surface of the towel ball (Fig. 4).

Fig. 3.

Fig. 4.

Fig. 3.—Correct. With towel gathered together, both hands and the distal two-thirds of both forearms are dried.

Fig. 4.—Correct. With the towel balled and held with the fingers of right hand, the left elbow is mopped; the towel ball is held on same surface by the fingers of the left hand to mop the right elbow.

By handling the towel in this manner there is no hanging end to come into contact with the shirt front, both hands and forearms are dried before any portion of the towel comes into contact with either elbow, and the thickness of the towel ball prevents elbow drippings from passing through to the hand.

Tears and punctures in the gloves, through which perspiration from the hand can escape into the operative field, occur with sufficient frequency to warrant their being seriously considered as a source of contamination. To guard against the development of infection from this source, not only must the hands and forearms be properly scrubbed, but they must also be protected from contamination during the process of drying.

Pediatric Surgery

CONDUCTED BY MARK M. RAVITCH, M.D.

DIARRHEA FOLLOWING RECTOSIGMOIDECTOMY FOR HIRSCHSPRUNG'S DISEASE

ORVAR SWENSON, M.D., JOHN H. FISHER, M.D., AND
JOHN E. S. SCOTT, M.A., BOSTON, MASS.

(From the Boston Floating Hospital)

WITH good surgical technique the postoperative complications of rectosigmoidectomy for Hirschsprung's disease are few and the results are satisfactory. The majority of patients have a smooth postoperative course and develop normal bowel function without incontinence. A few patients, however, suffer from a condition characterized by episodes of abdominal distention due to fecal and gas retention which is relieved by bouts of explosive liquid diarrhea occurring either spontaneously or after the passage into the rectum of a finger or a rectal tube. Such episodes may be accompanied by high fever and vomiting with severe loss of weight and become chronic if not treated promptly.

Most authors who have reported long-term follow-up studies mention these cases but do not discuss their management or the possible etiology. For example, Swenson[5] mentioned 5 cases in a series of 200, and Wyllie,[6] 10 in a series of 152. Hiatt[3] reported 9 cases in his series of 63 and stated that no pathogens were detected in the stools. Some of his patients suffered so severely with abdominal distention, fever, and diarrhea that a transverse colostomy had to be performed to afford relief. Ferguson,[2] in a series of 13 rectosigmoidectomy patients followed postoperatively for "several months," had "some" who were suffering from episodes of diarrhea and abdominal distention. No mention is made of this complication in the series reported recently by Langer and Thomson.[4]

Forty-two patients with Hirschsprung's disease were treated at the Boston Floating Hospital between February, 1954, and February, 1959, and 3 developed recurrent postoperative diarrhea. This gives an incidence of approximately 7 per cent which corresponds with the experience of other authors. Though the complication arises infrequently, it resists treatment and causes great distress for all concerned. It therefore merits further attention.

Hirschsprung's disease is caused by a lack of normal ganglia in the myenteric plexuses of the colon extending from the anorectal junction proximally for a variable distance. Since the internal anal sphincter is a condensation of the circular muscle of the rectum, it is also aganglionic and subject to the same

Received for publication Feb. 8, 1960.

functional disturbances as other parts of the colon. This may account for the outstanding feature of postrectosigmoidectomy diarrhea which is extreme anal spasm.

Failure of the anal sphincter to relax causes retention of feces and flatus with colonic distention and reduced water absorption.[1] Peristalsis increases until the resistance of the anal sphincter is overcome when the colonic contents are ejected under pressure causing uncontrollable diarrhea and incontinence. Similar explosions occur when the sphincter is forcibly opened with a finger or rectal tube. The fact that only a few patients are affected in this manner cannot be explained.

Conservative treatment which is frequently successful consists of decompression of the colon with a large rectal tube and gentle irrigations. There is usually a profuse discharge of foul liquid and gas when the tube is passed. By advancing the tube to various levels and maintaining patency with small injections of warm saline and gentle abdominal massage, the whole colon may be emptied. Twice daily irrigations for 2 or 3 days often produce a return of normal bowel function. Parents should be warned that these episodes of diarrhea may occur, and they should be instructed in the correct use of a rectal tube.

With consideration of the underlying pathology, an attempt was made to relieve the condition in the 4 cases previously mentioned by dividing the internal anal sphincter. The standard linear anal sphincterotomy frequently used for chronic posterior anal fissure was performed.

CASE REPORTS

CASE 1.—A male child, aged 2 years, 2 months, underwent a rectosigmoidectomy for Hirschsprung's disease in December, 1954. Following the operation, episodes of explosive diarrhea developed with anorexia and vomiting. Frequent rectal irrigations were advised but the condition continued unabated during the succeeding 4 years. In May, 1959, the child was readmitted for the repair of a left inguinal hernia. It was noted that the weight was 75 pounds and that the anal sphincter was tight. Herniorrhaphy and anal sphincterotomy were performed at the same operation. Three months later there was considerable improvement with increased appetite and weight gain. Bowel function was not completely normal for episodes of diarrhea were still occurring, though with reduced frequency. Between the episodes the child was continent with normal daily bowel movements. A more radical division of the sphincter might have achieved complete cure.

CASE 2.—In December, 1955, 5 feet of ileum and the ascending colon was resected in an 11-month-old male child for a gangrenous intussusception caused by a Meckel's diverticulum. Postoperatively bowel movements were frequent, but this gradually altered to complete constipation by January, 1957. Rectal biopsy revealed the presence of Hirschsprung's disease and in March, 1957, rectosigmoidectomy was performed at another hospital. This was followed by constipation and abdominal distention with atacks of explosive fluid diarrhea. Frequently, relief was obtained only by the passage of a large rectal tube and irrigations. The child was admitted in November, 1958, weighing 29 pounds. Small intestinal function was investigated and when this was found to be normal, anal sphincterotomy was performed. Nine months later the child had gained 16 pounds and was having normal bowel movements.

CASE 3.—A male child, aged 4 years, 5 months, underwent a rectosigmoidectomy for Hirschsprung's disease in November, 1958. Shortly after his return home, frequent, foul-smelling, fluid diarrhea commenced with intermittent fever and loss of weight. Conservative

treatment brought no response. The child was readmitted in January, 1959, and on examination the anal sphincter was found to be tight. Each rectal examination resulted in the forcible evacuation of liquid feces and gas. The condition did not respond to daily rectal irrigations and anal sphincterotomy was therefore performed. Seven months later the child's general condition was improved with a weight gain of 16 pounds. Bowel movements were still fluid though occasional solid ones were now passed and there was better control. The tone of the anal sphincter was still excessive. A more radical sphincterotomy would probably have alleviated all symptoms.

CASE 4.—A 1-year-old male child had a rectosigmoidectomy for Hirschsprung's disease in September, 1957. In July, 1959, he was readmitted with a 1-month history of constipation, abdominal distention, and vomiting. Rectal examinations caused massive, forcible evacuations of gas and fluid feces. Daily rectal irrigations relieved the condition and the parents were advised to continue these at home. In March, 1959, the fever, constipation, abdominal distention, and vomiting recurred and the child was readmitted. On examination the anal sphincter was tight. Sphincterotomy was performed and spontaneous bowel movements commenced postoperatively. Five months later the child had put on weight and though there were still occasional days when explosive diarrhea occurred, bowel function was improved.

The aganglionic bowel in all these cases extended to the common site in the rectosigmoid region and in none of them were any unusual features encountered at operation or in the immediate postoperative course. Examination of the feces in all cases revealed no pathogens.

The onset of colonic retention and diarrhea varied from immediately after the child's return home to 9 months postoperatively. There was no factor common to each case to account for abnormal bowel function, apart from excessive tone in the anal sphincter. Although only 1 of the 4 patients reported upon here was cured, the remainder were improved. If a more radical sphincterotomy had been performed in these cases, the results would probably have been more satisfactory.

SUMMARY

Four cases of episodic diarrhea with abdominal distention following rectosigmoidectomy for Hirschsprung's disease are reported.

It is suggested that this disturbance in bowel function is caused by the aganglionic internal anal sphincter which fails to relax and allow the feces to pass normally. The patients were treated by anal sphincterotomy resulting in complete cure of 1 and improvement of the remainder. It is suggested that a radical operation is necessary to achieve better results.

REFERENCES

1. Blickenstaff, D. D., Backmann, D. M., Steinberg, M. E., and Youman, W. B.: Intestinal Absorption of Sodium Chloride Solution as Influenced by Intraluminal Pressure and Concentration, Am. J. Physiol. **168**: 303-310, 1952.
2. Ferguson, C. C.: Megacolon in Infants and Children, Canad. J. Surg. **1**: 100, 1958.
3. Hiatt, R. B.: A Further Description of the Pathologic Physiology of Congenital Megacolon and the Results of Surgical Treatment, Pediatrics **21**: 825-831, 1958.
4. Langer, B., and Thomson, S.: Hirschsprung's Disease. Nine Years' Experience at the Hospital for Sick Children, Toronto, Canad. J. Surg. **2**: 123, 1959.
5. Swenson, O.: Follow-up on 200 Patients Treated for Hirschsprung's Disease During a Ten-Year Period, Ann. Surg. **146**: 706-714, 1957.
6. Wyllie, G. G.: Treatment of Hirschsprung's Disease by Swenson's Operation, Lancet **1**: 850-855, 1957.

CONGENITAL OBSTRUCTING GOITER

GEORGE B. PACKARD, M.D., EDWIN T. WILLIAMS, M.D., AND
SEYMOUR E. WHEELOCK, M.D., DENVER, COLO.

(From the Surgical and Medical Services, Children's Hospital)

A CASE of congenital goiter from the Denver Children's Hospital is reported to illustrate the problems encountered when pressure causes severe respiratory obstruction. One case of nonobstructive goiter is also reported for comparison. In spite of the rarity of the suffocative type in North America, the subject has received considerable attention in the literature with regard to cause, pathologic findings, and treatment.

Congenital goiter was formerly rather common in endemic goiter areas, especially in Europe where wide experience has been reported[9, 13, 44, 45] including many cases of strangling obstruction. Fabre and Thévenot,[13] for example, in their comprehensive article, collected 130 case reports, mentioned descriptions given since 1840, told of many suffocative cases associated with high mortality in Switzerland, described the first resection (an isthmectomy by Malgaigne in 1851), and added 78 references. They stated that most of the goiters examined weighed from 20 to 30 grams and showed diffuse hyperplasia. In recent years, since the almost universal use of iodine prophylactically, the situation has completely changed.

The American experience, by contrast, is very limited. While congenital goiter has been reported in endemic goiter areas,[37] the great majority were not of alarming size or of the strangling type. Today, all forms are uncommon in this country since iodized salt has come into common use, but congenital obstructive goiter does occur sporadically, though rarely, and may be severe enough to endanger life. In the North American literature, we have been able to find reports of 31 cases in which alarming suffocative symtoms were present. Treatment included both medical and surgical measures.

ETIOLOGY

No common mechanism was found to explain the extreme thyroid enlargement in the 31 cases. Goiters were observed in infants born of normal mothers, of mothers suspected to be hyperthyroid or hypothyroid, of those who had received thiouracil drugs or iodides, and of those who had not been given any known goitrogens. Of the 31 mothers, 8 had been on heavy iodide therapy throughout pregnancy, 3 on a combination of iodides and thiouracil, and 1 on thiouracil. Two mothers were considered myxedematous and 1 other was being treated for asthma with an unknown drug. Seven apparently were receiving no drugs. Unfortunately, in 9 cases there was no mention of maternal therapy. Though no common factor in etiology can be found in these 31 cases, it may be

Received for publication Feb. 11, 1960.

significant that 12 of the 22 mothers for whom treatment is recorded were on some thyroid-blocking therapy. The impact of maternal iodide or thiouracil feeding on the thyroid gland has been the subject of many studies. These describe animal and in vitro experiments as well as clinical experience in humans.

It has been demonstrated that if the serum iodide remains below a certain level, the thyroid accumulates iodine to form thyroid hormone, but when the serum iodide remains long above this critical point, the release of thyroid hormone from the gland may be almost completely inhibited.[40, 47] As a practical application of these conclusions, it is generally agreed[2, 26] that the ingestion of minute amounts of iodine, namely 0.1 mg. per day, prevents endemic goiter. However, many investigators have shown that prolonged ingestion of large amounts of iodine sets up a mechanism preventing release of thyroid hormone from the gland.[8, 14, 16] The action of thiourea derivatives may have the same practical effect by preventing synthesis of thyroid hormone.[1, 6, 12, 19] Most investigators[4, 6, 15, 19] agree that iodides, thiouracil, and thyroid hormone cross the placenta, but that thyroid-stimulating hormone does not.[1, 28, 31]

In the fetus, thyroid function is said to start at 14 weeks.[1] It is logical that, if the mother has a normal circulating amount of thyroid hormone, the fetus should receive no stimulus toward abnormal growth of the thyroid gland. On the contrary, if the fetus receives a subnormal amount of thyroid hormone into its circulation, it may be postulated that an increased output of thyroid-stimulating hormone from the fetal pituitary results in consequent hyperplasia of the thyroid.[1, 12, 39] On the same basis, it may be postulated that the transmission of excessive iodide or other blocking agent may in itself prevent fetal hormone production with consequent fetal pituitary stimulation toward thyroid hyperplasia.[4, 6, 28] These theories are based, however, on the unproved premise that this pituitary activity starts early in fetal life, since no maternal thyroid-stimulating hormone crosses the placenta.[1, 28, 31]

PATHOLOGY

In the 31 cases of severe obstructing goiter found in the North American literature, the pathologic findings were remarkably constant and quite generally support the theories already expressed. In 10 cases of thyroid resection, 8 were reported as parenchymatous or hyperplastic goiter[4, 7, 9, 11, 17, 35, 46] and 2 were described as Hürthle cell tumors.[27, 42] In 13 other cases which came to autopsy, 10 were considered typical hyperplastic goiters,[4, 11, 18, 21, 22, 24, 25, 36, 39] 1 was reported as a diffuse colloid goiter,[32] and in 2, the pathologic condition was not stated.[38] The great majority, then, of the congenital obstructing goiters where pathologic examination was possible were classified as the hyperplastic type. These were characterized by symmetrical enlargement and microscopically by follicles of varying size lined by cuboidal to columnar epithelium not infrequently infolded, with the lumens containing little or no colloid.

TREATMENT

In the older literature, which is predominantly Continental, operative treatment is not very strongly stressed. In the 31 cases of obstructive goiter

found in the American literature, 13 patients were treated by operation. Three of these had emergency tracheotomy without thyroid resection,[22, 24, 36] and all died. Ten underwent thyroid resection of varying amounts, and all recovered[4, 7, 9, 11, 17, 27, 35, 42, 46] with relief of the obstructive symptoms. Some operations were required for impending suffocation in the first few hours or days of life, while some resections were not performed for several weeks or months when persistence of obstructive signs demanded relief. The amounts of thyroid tissue removed ranged from 6 to 65 grams, a size quite in contrast to the normal neonatal total weight of 2 to 5 grams.[33]

Eighteen patients were treated expectantly or medically. It is not clear that all of these goiters should be classified as suffocative. Ten patients died, most of them from asphyxia, some too soon to permit much definitive treatment,[3, 21, 32, 37] but others at ages ranging from 2 days to 6 months.[11, 18, 21, 25, 37, 39] In some, operation seemed contraindicated and death was not clearly related to thyroid pressure in all instances. Expectant or medical treatment was adequate in other cases.[1, 4, 29, 30, 38, 41] It included the positioning of the head in hyperextension to permit better air intake, administration of oxygen, iodine given orally or by inunction, and feeding of thyroid extract or its hormone to reduce pituitary stimulation. These measures were intended to maintain aeration of the lungs pending goiter regression and lessened tracheal pressure, which often occurred spontaneously or with the help of thyroid feeding. One author[39] stated that if the baby could survive his first week, his chances were good. There were some exceptions.

Twenty-one other cases of congenital goiter were found in the American literature in which the thyroid was apparently not of sufficient size to cause alarming pressure.[5, 20, 23, 31, 34, 37, 43] The course in most of these cases was gradual subsidence or disappearance of the goiter. Obviously there must be a great many others not reported.

Two cases are reported here. One demonstrates the large goiter that spontaneously regresses and the other the suffocative type demanding thyroid resection.

CASE REPORTS

CASE 1.—D. L. K., the first male child of a farm family, was admitted to the Children's Hospital on Nov. 21, 1951, 3 days after delivery by cesarean section. His mother had always had an enlargement of the neck which had increased during pregnancy. She was asthmatic and had been taking iodine, but the dose and duration were not recorded. The baby weighed 6 pounds, 1 ounce (2.8 kilograms), looked well, and had a good cry. A large, symmetrical goiter filled his neck but no obstruction to breathing was evident though some difficulty in swallowing was noted. Since he seemed at ease and no further obstructing signs developed, he was released to his home 4 days later. His family doctor in 1957 wrote that the child appeared normal and healthy without signs of hypo- or hyperthyroidism. No mention was made of goiter.

CASE 2.—S. D. E., a male baby, was referred to the Children's Hospital on July 5, 1959, at 30 hours of age because of increasing difficulty in breathing, apparently caused by pressure from a large mass in the neck. He weighed 8½ pounds (3.85 kilograms), and was the third baby of the family. The neck enlargement was noted at birth and though it was said that he was in no respiratory distress, he had been receiving oxygen up to the time of admission.

The mother's prenatal course had been uncomplicated except that she suffered from chronic asthma for which she took regularly a saturated solution of potassium iodide (24 drops a day), as well as 1 other medicine. Her asthma had developed 2 years after her preceding pregnancy. When she was seen 1 week after the delivery, she appeared euthyroid. She had a small, symmetrical goiter which she said was long standing. Her mother in Germany had also had a goiter.

At admission, the baby was lying on his left side with his head in extreme hyperextension (Fig. 1). When quiet, his face appeared full and of normal color. Any attempt to change this position was met with active resistance. When he tried to cry, his color changed to a dusky cyanosis. A huge, collarlike mass filled the upper neck. It was symmetrical, with a thick central bridge connecting the 2 large lateral portions. It felt cystic. Except for these local findings, no particular abnormality was noted other than a high-pitched, systolic murmur at the apex. The hemoglobin estimation was 20.1 Gm. and the white blood count 21,500. The serum protein-bound iodine on the following morning was 16 mcg. per cent, the inorganic iodine over 500 mcg. per cent. (According to Danowski,[10] the normal protein-bound iodine in a 3-day-old baby averages 12.0 mcg.).

Fig. 1.—Baby at age 2 days. Neck is full of thyroid gland.

On the basis that some tumor subsidence might be expected, it was decided that the infant should be observed until the following day. Oxygen was administered continuously and pads were arranged to allow full hyperextension of the head. This decision might well have been a tragic one since twice during the night the baby had alarming periods of dyspnea and cyanosis which were relieved by suction and repositioning. On the following morning, July 6, no improvement could be recognized, but instead there was a greater respiratory difficulty with definite cyanosis. Surgical intervention was considered imperative to relieve the tracheal obstruction. With the use of endotracheal ether-oxygen anesthesia, a transverse incision was made which exposed a huge thyroid gland. Division of the isthmus and removal of the pyramidal lobe uncovered the trachea, but the lateral lobes looked so large and extended so far backward that it was feared this release might be inadequate. An estimated three-fourths of the left lobe, the isthmus, and an anterior slice of the right lobe were removed, with a combined weight of 21 grams. Following the operation, the baby appeared relieved and respiration seemed satisfactory.

The next day, July 7, the baby again did not seem to be breathing easily. By evening, his head was still held in a hyperextended position and respiratory effort increased as the hours passed. On direct visualization, the vocal cords were seen to move normally, but there was a surprising bulge of the posterior wall of the hypopharynx, as though from a forward-pressing mass definitely obstructing free air passage. On the following morning, July 8, again with intratracheal anesthesia, the operative wound was reopened for a tracheostomy as a final resort. Contrary to expectation, the trachea was no longer in the midline where it had been left at the first operation but was now displaced far to the left by the right lobe of the thyroid which occupied all the visible field. It was now realized that this lobe also extended far posteriorly and was responsible for the forward bulge of the hypopharynx. An estimated three-fourths of the lobe was resected (13 grams), allowing the trachea to regain its natural position. After consultation, and with realization that the baby had had 2 tracheal exposures and 2 tracheal intubations, it was decided that tracheostomy might be safer than risking laryngeal edema, and a No. 0 tube was inserted through a small incision in the trachea. The baby tolerated the procedure well, and his respiration and color were good postoperatively.

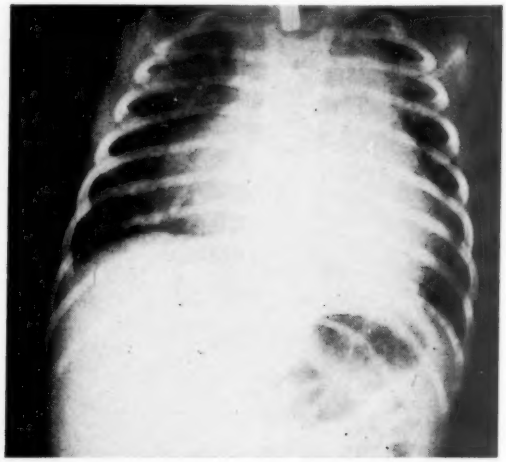


Fig. 2.—This film shows the heart nearly filling the left chest apparently from decompensation. Age 9 days.

In the following anxious days of posttracheostomy care with the small tube and trachea, edema and secretions tended to obstruct them. Complications were a tremendously enlarged heart (Fig. 2), diffuse moisture in the lungs, generalized edema, and a low liver. Very limited intravenous fluids, antibiotics, thyroid extract, digitalis, and on one occasion a mercurial diuretic seemed to be factors in relief. A complicated congenital heart anomaly was considered possible for a time, rather than a simple dilatation from failure. Feeding was mostly by gavage. Constant attention by special nursing was required to keep the small tracheostomy tube clear and it was not until removal of the tube on July 18 that breathing was improved and nourishment was taken in proper amounts. At that time, direct laryngoscopy showed normal cord movement and almost complete lack of edema. In retrospect, earlier tube removal would have been preferable. The child was dismissed on July 25, he was gaining weight, had a good cry, sucked well, was active, and had a good color.

Pathologic findings were reported by Dr. E. C. Beatty, Jr., as showing a total thyroid weight of 34 grams. Cutting revealed a tan-red, homogeneous, meaty surface and a soft, rubbery consistency. Sections were described as showing extreme hypercellularity with numerous acini of varied sizes, containing little or no colloid and lined with tall cuboidal or low columnar cells. The epithelium at some places was thrown into folds. The diagnosis was diffuse hyperplasia of the thyroid (Fig. 3).

The baby and mother returned for a check examination on Oct. 2, 1959. The baby appeared well and active, perhaps a little thin (Fig. 4). His cry seemed somewhat hoarse, though his parents thought it not different from that of their other children. His weight

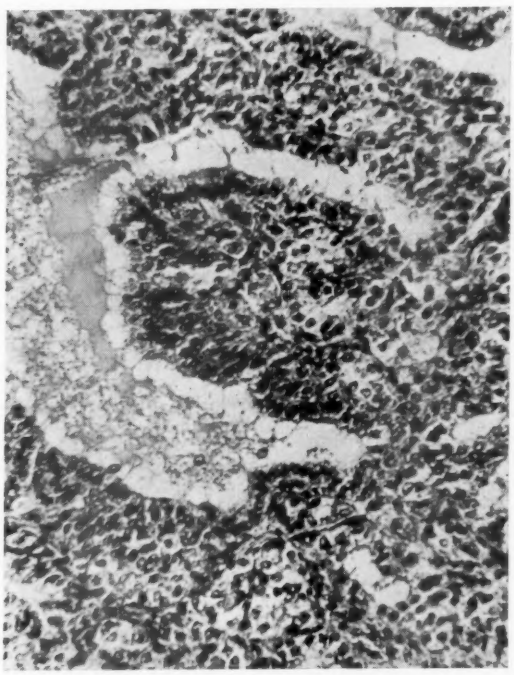


Fig. 3.—Photomicrograph of tissue removed at thyroid resection shows epithelial hyperplasia, papillations, and reduced colloid with peripheral vacuolization. (Hematoxylin and eosin; ×120, reduced ⅕.)

Fig. 4.—Appearance of baby at 3 months. No neck swelling.

was 10 pounds (4.5 kilograms), his length was normal for his age, and there were no heart murmurs and no râles. The electrocardiogram was summarized as sinus tachycardia. The protein-bound iodine was 7.1 mcg. and serum cholesterol 169 mg. per cent. X-ray examination of the chest (Fig. 5) showed the heart smaller than in July but still moderately enlarged with no specific contour, and the lungs clear. In films of the extremities, bone age was considered normal for 3 months.

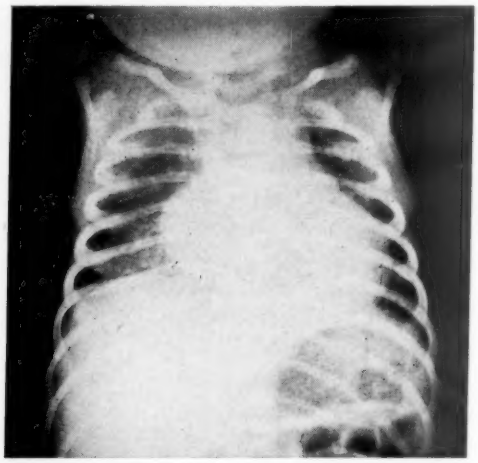


Fig. 5.—Chest film at 3 months. Heart much smaller though considered moderately enlarged.

The mother appeared as before, slender, of the inactive type, with a moderate symmetrical enlargement of the thyroid. She was still taking her asthma medicine, her protein-bound iodine registered 16.0 mcg. per cent, the inorganic serum iodine above normal, and her serum cholesterol as 279 mg. per cent.

DISCUSSION

Apparently most congenital goiters are relatively harmless and disappear with medical or expectant care. These cases are reported to show a contrast and emphasize that a serious emergency may develop when a congenital goiter is large enough or wedged in a position to cause tracheal obstruction. In most cases, the medical attendants have had no experience with expected course or prognosis to guide them toward the proper treatment. Hill,[18] in 1933, reporting a death from choking by goiter in a 4-day-old infant, stated that this was the first such case recorded in 12,908 deliveries in the obstetric department of the White Memorial Hospital in Los Angeles. Handelsman,[17] in 1947, reported a thyroidectomy in a 6-day-old child and could find no other case reported at the Baltimore Sinai Hospital or at The Johns Hopkins Hospital. At the Denver Children's Hospital, of 146,903 admissions in all service branches since 1942, only the 2 cases of congenital goiter here reported appear in the diagnosis files.

In our second case, where it was soon apparent that suffocation was impending and required removal of the obstructing mass for relief, the contemplated 24 hour observation actually jeopardized the outcome. At operation, which seemed at the time fully adequate, the potential danger of leaving most of one lobe was not appreciated. The unforeseen pressure of the residual portion on the posterior pharyngeal wall nearly resulted in treatment failure. The need for tracheostomy may well be questioned in spite of the possibility of

laryngeal edema in a twice-exposed and twice-intubated trachea. The universally bad results from this procedure in previously recorded cases were carefully considered but it was also realized that a hasty tracheostomy in a short neck bulging with goiter was not exactly comparable to the operative exposure here. Regardless of conditions, however, a tube which is necessarily small and unavoidably irritating to the trachea allows barely sufficient air intake. Recovery in this case is, of course, the result of relief of tracheal obstruction but successful result would not have been possible without continuous close and expert nursing care as well as advice and help from the medical department.

From the experience in this obstructive case and in the other similar cases reported, it is apparent that partial and probably symmetrical thyroidectomy is the treatment of choice in suffocative goiter. Deliberate technique is permitted under an anesthetic administered through an intratracheal tube, the area of operative exposure is not large, and control of bleeding is no more difficult than elsewhere. All of these factors have contributed to the universal success of the procedure. When suffocation appears impending, there is certainly no reason for delay and no place for watchful waiting. Unfortunately, in some cases, the complete blockage of air intake immediately at birth did not allow any deliberate operative procedure.

SUMMARY

Congenital goiter, not uncommon before the general use of prophylactic iodine ingestion, is seldom seen today and the suffocative type is extremely rare.

Nearly all neonatal goiters reported in this country are of the hyperplastic variety. While the mechanism of their formation is not by any means clearly explained in all cases, evidence suggests that deficient thyroid hormone in the fetus has activated the pituitary toward stimulating growth of the thyroid gland. Maternal hypothyroidism, often the result of heavy iodide or of thiouracil therapy, seems to have been the predisposing factor in many cases.

A case of severe obstructing goiter treated by resection, with eventual recovery in spite of a supplementary tracheotomy, is reported as an addition to 10 successful cases previously described.

The necessity for prompt resection in suffocative cases is emphasized since tracheotomy alone has been universally unsuccessful. Medical or expectant care has been associated with a high mortality in contrast to the excellent results following partial thyroidectomy.

The author wishes to thank Dr. Dalton Jenkins of the University of Colorado Department of Medicine for his review and suggestions concerning the endocrinologic aspects of the cases.

REFERENCES

1. Aaron, H. H., Schneierson, S. J., and Siegel, E.: Goiter in Newborn Infant Due to Mother's Ingestion of Propylthiouracil, J. A. M. A. 159: 848-850, 1955.
2. Bell, G. O.: Prolonged Administration of Iodine in the Pathogenesis of Simple Goiter and Myxedema, Tr. Am. Goiter A., 1952, 28-37, 1953.
3. Bernhard, W. G., Grubin, H., and Scheller, G. A.: Congenital Goiter, A. M. A. Arch. Path. 60: 635-638, 1955.
4. Bongiovanni, A. M., Eberlein, W. R., Thomas, P. Z., and Anderson, W. B.: Sporadic Goiter of the Newborn, J. Clin. Endocrinol. 16: 146-152, 1956.

5. Branch, L. K., and Tuthill, S. W.: Goiters in Twins Resulting From Propylthiouracil Given During Pregnancy, Ann. Int. Med. **46**: 145-148, 1957.
6. Caren, R.: The Goitrogens in Thyrotoxicosis Complicating Pregnancy, J. Clin. Endocrinol. **9**: 903-909, 1949.
7. Cauble, W. G., and Lockhart, J. G.: Congenital Goiter: Report of Two Cases, Am. Surgeon **24**: 118-122, 1958.
8. Childs, D. S., Jr., Keating, F. R., Jr., Rall, J. E., Williams, M. M. D., and Power, M. H.: The Effect of Varying Quantities of Inorganic Iodide (Carrier) on the Urinary Excretion and Thyroidal Accumulation of Radioiodine in Exophthalmic Goiter, J. Clin. Invest. **29**: 720-738, 1950.
9. D'Abreu, A. L., and Wood, B. S. B.: A Case of Sporadic Congenital Goiter, Arch. Dis. Childhood **25**: 235-239, 1950.
10. Danowski, T. S., Johnston, S. Y., Price, W. C., McKelvey, M., Stevenson, S. S., and McCluskey, E. R.: Protein-bound Iodine in Infants From Birth to One Year of Age, Pediatrics **7**: 240-244, 1951.
11. Davies, J. R.: Sporadic Congenital Obstructive Goiter With Recovery Following Operation in a Thirteen-day-old Infant, J. Pediat. **22**: 570-580, 1943.
12. Elphinstone, N.: Thiouracil in Pregnancy, Lancet **1**: 1281-1283, 1953.
13. Fabre, J., and Thévenot, L.: Le Goitre chez le nouveau-né, Rev. de chir. **37**: 781-805, 1908.
14. Fawcett, D. M., and Kirkwood, S.: The Mechanism of the Anti-Thyroid Action of Iodide Ion and of the Aromatic Thyroid Inhibitors, J. Biol. Chem. **204**: 787-796, 1953.
15. Freiesleben, E., and Kjerulf-Jensen, K.: The Effect of Thiouracil Derivatives on Fetuses and Infants, J. Clin. Endocrinol. **7**: 47-51, 1947.
16. Goldsmith, R. E., and Eisele, M. L.: The Effect of Iodide on the Release of Thyroid Hormone in Hyperthyroidism, J. Clin. Endocrinol. **16**: 130-137, 1956.
17. Handelsman, J. C., and Sussman, H.: Suffocative Goiter in Newborn, Ann. Surg. **145**: 108-114, 1947.
18. Hill, A. L.: Congenital Goiter, Arch. Pediat. **50**: 424-430, 1933.
19. Hughes, A. M.: Cretinism in Rats Induced by Thiouracil, Endocrinology **34**: 68-76, 1944.
20. Javett, S. N., Senior, B., Brando, J. L., and Heymann, S.: Neonatal Thyrotoxicosis, Pediatrics **24**: 65-73, 1959.
21. Jones, D. B.: Congenital Goiter in North America, Am. J. Path. **27**: 85-93, 1951.
22. Kunstadter, R. H.: Congenital Goiter Causing Death of a Newborn Infant, J. Pediat. **32**: 711-714, 1948.
23. Lincoln, A. E.: Congenital Hyperplasia of the Thyroid Gland, Am. J. Clin. Path. **23**: 1019-1023, 1953.
24. Mitchell, D. S., and Struthers, R. R.: Congenital Hypertrophy of the Thyroid Gland with Report of a Case, Canad. M. A. J. **28**: 27-30, 1933.
25. Moran, T. J.: Congenital Goiter With Death from Milk-Aspiration Pneumonia, Arch. Pediat. **54**: 213-219, 1952.
26. Morgans, M. E., and Trotter, W. R.: Myxedema Accredited to Iodide Administration, Lancet **2**: 1335-1337, 1953.
27. Morrow, W. J.: Hürthle Cell Tumor of the Thyroid Gland in an Infant, Arch. Path. **40**: 387-391, 1945.
28. Nikitovich, M., and Knobil, E.: Placental Transfer of Thyrotropic Hormone in the Rat, J. Clin. Endocrinol. **15**: 837-838, 1955.
29. Parmalee, A. H., Allen, E., Stein, I. F., and Buxbaum, H.: Three Cases of Congenital Goiter, Am. J. Obst. & Gynec. **40**: 145-147, 1940.
30. Pearlman, L. N.: Goitre in a Premature Infant, Canad. M. A. J. **70**: 317-318, 1954.
31. Peterson, R. R., and Young, W. C.: The Problem of Placental Permeability for Thyrotropin, Propylthiouracil and Thyroxine in the Guinea Pig, Endocrinology **50**: 218-225, 1952.
32. Petty, C. S., and Di Benedetto, Robert L.: Goiter of the Newborn, New England J. Med. **256**: 1103-1105, 1957.
33. Potter, E. L.: Pathology of the Fetus and Newborn, Chicago, 1952, The Year Book Publishers, Inc., pp. 276-279.
34. Pugh, W. C.: Diffuse Nontoxic Goiter as a Cause of Persistent Face Presentation, Am. J. Obst. & Gynec. **66**: 688-689, 1953.
35. Schifrin, N., and Hurwitt, E. S.: Hypothyroidism Following Thyroidectomy for Congenital Obstructive Goiter, J. Pediat. **39**: 597-605, 1951.
36. Seligman, B., and Pescovitz, H.: Suffocative Goiter in Newborn Infant, New York J. Med. **50**: 1845-1847, 1950.
37. Skinner, H. H.: Congenital Goiter, J. A. M. A. **82**: 1190-1192, 1924.
38. Skinner, H. H.: Results in Congenital Goiter, M. J. & Rec. **127**: 381-383, 1928.

39. Solis-Cohen, L., and Steinbach, M.: Sporadic Congenital Goiter Causing Death in an Infant, Am. J. Dis. Child. **58**: 1067-1071, 1939.
40. Stanley, M. M.: The Effect of Iodide Ion on Thyroid Hormone Formation in Man, J. Clin. Invest. **28**: 812-813, 1949.
41. Sweet, P. W.: Goiter in Newborn Infant, Northwest Med. **27**: 183-184, 1928.
42. Symmers, D.: Congenital Hürthle Cell Tumor, Arch. Path. **31**: 99-102, 1941.
43. Warren, S., and Shpiner, L. B.: Primary Hyperplasia of the Thyroid in One of Still-Born Twins, J. A. M. A. **109**: 575-577, 1937.
44. Wegelin: Report of Switzerland Goiter Commission, 1922, in Wieland.[45]
45. Wieland, E.: Ueber Struma congenita, Schweitz. Med. Wchnschr. **5**: 850-852, 1927.
46. Williamson, G. R.: Congenital Goiter, J. Pediat. **2**: 458-462, 1933.
47. Wolff, J., and Chaikoff, I. L.: Plasma Inorganic Iodide as a Homeostatic Regulator of Thyroid Function, J. Biol. Chem. **174**: 555-564, 1948.

Recent Advances in Surgery

CONDUCTED BY ALFRED BLALOCK, M.D.

PROFOUND HYPOTHERMIA COMBINED WITH EXTRACORPOREAL CIRCULATION FOR OPEN HEART SURGERY

WILL C. SEALY, W. GLENN YOUNG, JR., IVAN W. BROWN, JR., WIRT W. SMITH, AND ALAN M. LESAGE, DURHAM, N. C.

(From the Department of Surgery, Division of Thoracic Surgery, Duke University Medical Center)

INCREASING experience with various techniques of complete cardiopulmonary bypass makes evident the fact that there are now several extracorporeal systems adequate for clinical use. Unfortunately some are so complicated that they have associated with them certain technical hazards that can be overcome only by the use of several highly skilled human monitors. This complexity is fostered by the high flow rates required by normal body temperature.

In searching for a technique using a low flow system, we first employed in clinical practice in 1956[1] the combination of hypothermia and extracorporeal circulation. Some of the early workers[2] in this field warned that the 2 techniques combined were fraught with unusual hazards. It was postulated that the heart in particular might be irreversibly damaged by the cold. Our first trials with external cooling were laborious but demonstrated the safety afforded by these 2 techniques. It soon became evident, confirming our laboratory studies, that the heart (second only to the brain) benefited most by this combination.[3] With the introduction of core cooling by means of a heat exchanger[4] in the pump-oxygenator system, the clinical application became practical and hypothermia could for the first time be safely extended to profound levels. This temperature level has many advantages. It permits further reduction in flow rates, longer safe operating time with these low flow rates, periods of circulatory standstill when certain technical situations demand it, and, as we first demonstrated,[5] allows long periods of cardiac anoxia.

This paper will relate our laboratory and clinical experiences with hypothermia of 20° C. or lower for cardiopulmonary bypass.

CONTROL OF THE TEMPERATURE

Temperatures of 3 to 37° C. were induced, controlled, and reverted by a heat exchanger incorporated in the arterial side of a DeWall-Lillehei pump-

Suported in part by Research Grant H-1782 C4, National Institutes of Health, and the Duke University Regional Center for the Study of Aging.

Received for publication Feb. 11, 1960.

oxygenator. The flow was controlled by regulation of the arterial input, while the venous return was pumped back to the oxygenator. Partial body perfusion for cooling and warming with flow rates of 15 to 50 c.c. per kilogram per minute, depending on weight, was used until the heart slowed and lost its effective action or until occlusion of the venae cavae occurred. Then, with the temperature at the desired level, complete bypass using the same flow rates was employed. In certain of the animal experiments when the chest was not opened, the flows were reduced from 50 to 25 c.c. per kilogram per minute when cardiac action became ineffective.

EXPERIMENTAL STUDIES

With hypothermia induced and controlled by a heat exchanger pump-oxygenator system, a series of experiments were performed to determine (1) the tolerance of animals to temperatures below 20° C., (2) the incidence of ventricular fibrillation and its control with quinidine, (3) the tolerance of the cold heart to anoxia, and (4) the safe period of complete circulatory standstill at temperatures below 20° C.

In the first study,[5] 22 dogs were cooled by perfusion to a range of 10 to 20° C. as recorded in the esophagus. The heart was placed in standstill with potassium-magnesium-Prostigmine solution when the heart temperature reached 28 to 30° C. In 11 dogs the pump was stopped for periods of time from 10 to 30 minutes, thus producing complete circulatory standstill. Five of the 11 dogs did not survive. In a second group of 11 dogs, the oxygenator was by-passed for an average of 25 minutes (variation from 15 to 30 minutes), but perfusion was continued, which was with venous blood since the blood did not pass through the oxygenator. Three dogs in this group did not survive. Only 2 dogs in this study were perfused at temperatures below 13° C., the majority being above 15° C. In group 2, it was noted that at these temperatures oxygen was still being taken up, as shown by the steady decrease in the oxygen saturation of the perfused blood.

In the next series,[6] which consisted of 18 dogs, the animals were cooled but the chest was not opened and chemical cardioplegia was avoided. Periods of standstill of 30 minutes were permitted in 2 dogs, 1 hour in 10 dogs, 1½ hours in 6 dogs, 2 hours in 2 dogs, and 3 hours in 2 dogs. All of the dogs in complete circulatory standstill for 30 minutes and 1 hour survived. Four of the 6 dogs subjected to 90 minutes of standstill lived, but all of those remaining died. The electroencephalogram became silent at 16 to 20° C., while the heart went into standstill at temperatures from 13 to 9° C. The surviving animals were all normal.

To prevent ventricular fibrillation since the chest was not opened, quinidine hydrochloride, 28 to 30 mg. per kilogram, was given intravenously just before the start of the perfusion. The dosage was regulated by the appearance of electrocardiogram changes denoting early quinidine effect. This prevented fibrillation in every instance, provided circulatory standstill did not exceed 1 hour.

In a series of acute experiments with the dogs perfused in exactly the same way but not given quinidine, it was noted that ventricular fibrillation occurred either during warming or cooling in every instance, usually at temperatures of 15 to 18° C.

In these experiments coronary artery occlusion existed, since the heart was in standstill and the pump was stopped. Cardiac function returned in all dogs after 1 hour of circulatory arrest. At 1½ hours, 4 of 6 dogs had fibrillation, while circulatory arrest above this time resulted in ventricular fibrillation in all. Even though the temperature of the dog was greatly reduced, an oxygen debt occurred. This is shown in the reduction of the oxygen content of the mixed venous blood to levels of 70 to 80 per cent after 1 hour of arrest as indicated by the first minute sample taken after resumption of perfusion. The oxygen debt incurred may also have taken place in the heart, which means that prolonged periods of anoxia in the cold heart can result in damage. In the earlier experiments where cardiac standstill was induced by potassium, magnesium, and Prostigmine, there was a poorer survival rate, though of course the animals had been subjected to an open thorax and the actual heart temperatures were above 25° C.

From these studies we concluded that cold per se was not harmful and that its effect could always be reversed. Periods of complete cessation of circulation would be possible for periods up to 1 hour at temperatures in the esophagus of about 10° C. Ventricular fibrillation could be prevented by quinidine, obviating the need of the open chest and permitting the use of this technique in other surgical procedures. With the chest open quinidine has not been needed, for electric shock was always successful. The upper limits of tolerance of the cold dog heart to circulatory occlusion is about 1 hour, which may be the limiting factor to generalized circulatory arrest. Other studies which are not outlined in detail confirm the fact that oxygen consumption at 20° C. is 20 to 25 per cent of normal, while at 10° C. it may be 10 per cent of normal. When an oxygen debt occurs during hypothermia, the dog is capable of increasing the arteriovenous oxygen difference to compensate for it.

CLINICAL OBSERVATIONS

The clinical material reviewed consists of 33 patients submitted to operation for a variety of intracardiac defects plus 3 patients who were operated upon for serious neurologic diseases.

The lowest temperatures attained, as measured in the midesophagus, varied from 3 to 22° C. Perfusion was partial until either the heart action became ineffective or caval occlusion was carried out. Then the patient was for the first time on complete bypass. This technique permitted low extracorporeal perfusion rates throughout, for the total blood flow during the time metabolic needs were high due to high body temperature was the sum of the patient's own cardiac output plus the extracorporeal assistance. When complete bypass was needed, the metabolic needs, because of the hypothermia, were such that even low perfusion rates were adequate. With complete bypass, flow rates of 15 to 25 c.c. for patients above 30 kilograms in weight was used, while for those below this weight

from 35 to 50 c.c. was needed. The total perfusion rate never exceeded 2,400 c.c. per minute for any patient.

The estimate of the exact flow rate needed has been based entirely on laboratory and clinical experience. The most useful measure has been the venous oxygen saturation which we have sought to keep well above 70 per cent and the maintenance at all times of a normal arteriovenous difference. As shown in Fig. 1, a plot of the arteriovenous differences in all of our patients, regardless of the presence or absence of preoperative cyanosis, shows only 3 to 4 samples out of 60 are ever above 6 vol. per cent. This difference is actually decreased with the lowered temperature in spite of the fact that total perfusion rate of the body was greatly reduced when the patients were put on complete bypass. It has not been possible to be more accurate with estimation of the exact needs based on our clinical studies, for we have varied the flows greatly in order to permit better visualization of the defect or to reduce the damaging effect on the blood of passage of large amounts of it through a coronary sucker system. Fig. 2 demonstrates how the flow rates may be varied to expedite the operation. In

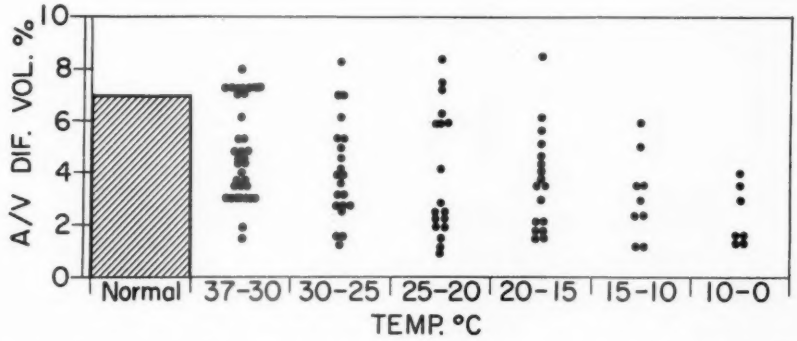


Fig. 1.—Graph showing the arteriovenous oxygen differences at various temperatures in 20 patients undergoing open heart operations. The solid block is the normal difference at 37° C. temperature. The remainder is our observation.

this instance there were 3 periods when the circulation was arrested completely for 3 minutes. Temperatures below 20° ought to permit useful periods of standstill on the basis of the observations on dogs and the clinical experiences with hypothermia only at 32 to 28° C.

In deciding exactly what temperature to use for a specific case, the only important consideration is the length of the operation. Since atrial secundum defects, for example, can be repaired in times less than 20 minutes, temperatures below 28° C. are not usually employed. For longer operations, because of increased safety and greater operative convenience, temperatures below 20° C. are used. Temperatures of 20° C. or lower are designated as profound hypothermia, for at this point metabolic changes of importance are noted. First, the oxygen needs of the body are reduced well below 25 per cent of normal at 20° C., but from this point on down the decrease in oxygen needs is not nearly so rapid. Second, the electroencephalogram may become silent, indicating nearly complete cessation of metabolic activity in the brain. This electroencephalographic change has been observed in all of our patients cooled below 20° C. The third

important change is noted in the heart. The heart action usually becomes ineffective somewhere between 25 and 20° C., and between 20 and 10° C. it will probably go either into standstill or into ventricular fibrillation and then standstill. Oxygen consumption studies indicate that the heart uses little oxygen at these levels.[7] These changes are all indicative of a greatly reduced metabolic rate and therefore a greater safety when periods of circulatory standstill are desired or when coronary artery flow arrest is needed.

The temperatures used in the heart patients have varied from 9 to 22° C. In cases of severe deformity we have used temperatures below 15° C., while for the milder ones the range of 20 to 18° C. is employed.

The use of cardiac arrest during open heart surgery has always been attractive since this expedites the intracardiac procedure and reduces the loss of blood from the heart that could be used to perfuse the remainder of the body. At normothermia and with high perfusion rates, this could be obtained with chemicals or anoxia, but only at the cost of anoxic heart damage and left heart distention with its cardiac and pulmonary sequelae. Our previous observations[8]

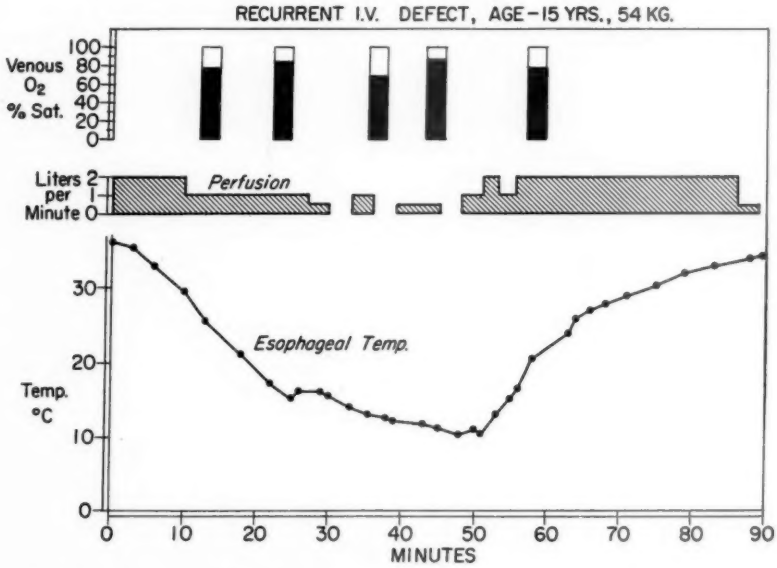


Fig. 2.—These are the data on a patient with a recurrent interventricular septal defect that was originally a part of a tetralogy of Fallot. Note the periods of complete circulatory standstill and its effect on the venous oxygen saturation.

have shown that the heart that is cold but arrested with potassium, magnesium, and Prostigmine will tolerate periods of arrest up to 60 minutes in dogs and 30 to 50 minutes in man. The low flows employed prevent the troublesome left heart distention.

We first demonstrated in clinical trials that profound hypothermia could be employed to either slow or arrest the heart's action yet permit long periods of cessation of coronary blood flow.[9] In our clinic this has been employed in 2 ways. The first and preferable way is by cooling the heart along with the remainder of the body. This, however, may increase the perfusion time in very large patients. It is our reasoning that if the heart is so responsive to the

beneficial action of cold, then the remainder of the body also needs this beneficial effect. The second technique is by selective perfusion of the heart with cold blood, reducing its temperature to below 20° C., but keeping the remainder of the body above 26 to 28° C. As we have pointed out, this permits continued perfusion of oxygenated blood though at rates so slow that the coronary venous drainage is small or, if necessary, the coronary perfusion can be stopped completely. This is easily accomplished by means of a leadoff from the arterial line passing through a second heat exchanger. The technique of cold-induced cardioplegia has been used in 25 patients. In 23 it was obtained as a part of general hypothermia, while in 2 this was done differentially by coronary perfusion. In 17 patients coronary occlusion was employed, while in the others coronary perfusion was continued during standstill. Ventricular fibrillation occurred either during cooling or rewarming in 18 patients, but 12 hearts went into standstill. In 10 of the 18, ventricular fibrillation continued but was easily reverted with electric shock. The remainder spontaneously reverted to normal rhythm. Perhaps it is not proper to use the term cardioplegia since the heart may beat occasionally or even fibrillate under these conditions. In the 3 neurosurgical operations, cardiac standstill did not occur until esophageal temperatures were reduced below 15° C. The important fact to be remembered is that cold is used primarily to increase the tolerance of the heart to coronary occlusion.

Perfusion techniques to control not only the temperature level but the distribution of cold are important in the employment of core cooling. Only a few of the important points can be covered here. This technique is not well suited for venous gravity drainage, since this would cool the heart very rapidly and prevent it from assisting in the maintenance of adequate circulation when the temperature of the entire body is still high. It is necessary to use venous outflows from the superior vena cava alone at the start of the partial perfusion in order to distribute the cold to the upper portion of the body. If the outflow is through both venous catheters at the beginning, then most of the cold will be distributed to the lower one-half of the body. On rewarming these same principles must be observed.

The time needed to reach and then revert the profound hypothermia does add to the total extracorporeal perfusion time. In this series the cooling and warming times were about 20 minutes each. During this time, some necessary technical procedure was being completed, so that the time was not wasted. During rewarming, the superior vena caval catheter was very frequently removed. The period of rewarming was begun as the heart was being closed and therefore much of the time was consumed by this maneuver. Temperatures of 34° C. in the esophagus were considered adequate on rewarming.

Results of operation in this group reveal a high mortality rate since most of the patients presented poor risks because of complicated anomalies. In the 15 patients with tetralogy of Fallot there were 4 deaths, while there was 1 death in the 5 with ventricular septal defects and none in 8 septum primum patients. The over-all mortality rate for the group was 30 per cent. In none could the deaths or complications be attributed to the perfusion or the profound hypothermia.

CONCLUSIONS

Profound hypothermia induced and controlled by core cooling has been found to be a safe technique for cardiopulmonary bypass and offers the following advantages: (1) A simple pump-oxygenator is used at low perfusion rates. (2) Small catheters are used for connection to the pump. (3) Periods of markedly reduced flow rates are possible for control of the blood loss from coronary sinus and bronchial artery return to the heart. (4) Periods of complete circulatory standstill are possible for difficult intracardiac maneuvers. (5) There is increased tolerance of the heart to anoxia and actual cold cardioplegia. (6) Extension of this technique can be made to other surgical fields.

REFERENCES

1. Sealy, Will C., Brown, Ivan W., Jr., Young, W. Glenn, Jr., Stephen, C. R., and Harris, J. S.: Hypothermia, Low Flow Extracorporeal Circulation and Controlled Cardiac Arrest for Open Heart Surgery, Surg. Gynec. & Obst. **104**: 441, 1957.
2. Lillehei, C. W., Warden, H. E., Varco, R. L., Stanley, P., and DeWall, R. A.: Use of Cardiopulmonary Bypass in Surgical Treatment of Congenital or Acquired Heart Disease, A. M. A. Arch. Surg. **75**: 928, 1957.
3. Sealy, Will C., Young, W. Glenn, Jr., Brown, Ivan W., Jr., Harris, J. S., and Merritt, D. H.: Potassium, Magnesium, and Neostigmine for Controlled Cardioplegia: A Report of Its Use in 34 Patients, J. Thoracic Surg. **37**: 655, 1959.
4. Brown, Ivan W., Jr., Smith, W. W., and Emmons, W. O.: An Efficient Blood Heat Exchanger for Use With Extracorporeal Circulation, SURGERY **44**: 372, 1958.
5. Lesage, Alan M., Sealy, W. C., Brown, Ivan W., Jr., and Young, W. Glenn, Jr.: Hypothermia and Extracorporeal Circulation: Experimental Studies on Profound Hypothermia of 10° C. to 20° C., A. M. A. Arch. Surg. **79**: 607, 1959.
6. Sealy, Will C., Johnson, Paul A., Lesage, Alan M., and Young, W. Glenn, Jr.: The Effect of Quinidine on Ventricular Fibrillation in Hypothermic Dogs, Proc. Soc. Exper. Biol. & Med. **101**: 456, 1959.
7. Young, W. Glenn, Jr., Sealy, Will C., Brown, Ivan W., Jr., Hewitt, W. C., Jr., Callaway, Henry A., Jr., Merritt, D. H., and Harris, J. S.: A Method for Controlled Cardiac Arrest as an Adjunct to Open Heart Surgery, J. Thoracic Surg. **32**: 604, 1956.
8. Sealy, Will C., Young, W. Glenn, Jr., Brown, Ivan W., Jr., Lesage, Alan M., Callaway, Henry A., Jr., Harris, J. S., and Merritt, D. H.: Potassium, Magnesium, and Neostigmine for Controlled Cardioplegia: Studies on the Dog Using Extracorporeal Circulation and Hypothermia, A. M. M. Arch. Surg. **77**: 33, 1958.
9. Young, W. Glenn, Jr., Sealy, W. C., Brown, Ivan W., Jr., Smith, W. W., Callaway, Henry A., Jr., and Harris, J. S.: Metabolic and Physiologic Observations on Patients Undergoing Extracorporeal Circulation in Conjunction With Hypothermia, SURGERY **46**: 175, 1959.

Surgical History

CONDUCTED BY JOHN R. PAINE, M.D.

THE OPERATIONS FOR INGUINAL HERNIA

I. BASSINI, HALSTED, ANDREWS, FERGUSON

MARK M. RAVITCH, M.D., AND JAMES M. HITZROT, II, M.D., BALTIMORE, MD.

(From the Departments of Surgery of The Johns Hopkins University School of Medicine, The Johns Hopkins Hospital and The Baltimore City Hospitals)

EDOARDO BASSINI*
1844—1924

Edoardo Bassini was born in Pavia, in Lombardy, on April, 14, 1844. He attended school in the town of his birth and received his doctorate in medicine from the University of Pavia at the early age of 22. The year of his graduation, 1866, also marked the unification of Italy. The liberation was largely a movement of the young intellectuals, and one in which young Bassini eagerly participated. Although a graduate in medicine, he chose to take part as a private in the ranks, and engaged in two campaigns in that capacity. In the second of these, the ill-fated attempt of Garibaldi to wrest Rome from the French and Papal forces, the Italians were hopelessly outnumbered, and were badly defeated. Bassini sustained a bayonet wound of the groin which festered and resulted in a fecal fistula. He was taken prisoner and remained in the hospital for a number of months. When eventually discharged, he returned to Pavia. The fistula reopened, and was treated, until definitively healed, by Luigi Porta, Chief of the University Surgical Clinic, who subsequently became Bassini's preceptor in surgery.

After his recovery, Bassini served as second assistant to Porta, and devoted himself particularly to the study of anatomy and surgical pathology. At this early period he made numerous dissections of the inguinal region, anticipating his future interest in the problem of hernia. To complete his studies, he later visited other famous clinics. He spent a year in Vienna with Billroth, worked with Langenbeck in Berlin, and with Lister and Spencer-Wells in London. He visited Paris too, but remained only a short time because of the bitterness which he still felt toward the French for their affiliation with the Pope during the War of Unification.

Received for publication Sept. 20, 1959.

*Sketch of Bassini's life from Leo Zimmerman and Richard Heller in *Scritti di Chirurgia Erniaria* by the Faculty of Medicine and Surgery of the University of Padua, vol. 1, p. 606, 1937, published by Tipografia del Seminario di Padova.

EDOARDO BASSINI

From the National Library of Medicine, Washington, D. C.

In 1874, at the age of 30, he was invited to return to Pavia as first assistant to Porta, and to teach in the University. The following year his Chief became ill, and Bassini acted in his stead. When Porta died, however, the Chair was awarded to someone else and, disappointed, Bassini resigned from the University and returned to London. Six months later he was called to Parma to teach clinical surgery. Here he introduced Listerian methods, one of the first to bring antiseptic surgery to Italy. Two years later he went to Spezia, as Head of the Department of Surgery there. In 1882 he was called to the University of Padua, to the Chair of Surgical Pathology. In this famous institution he spent the following 37 years of his life. He succeeded to the chair of clinical surgery in 1888, a position which he held until his resignation at the age of 75, in 1919. During this long and fruitful period, his epoch-making work on hernia appeared, as well as contributions to numerous other phases of surgical literature. He was the recipient of many honors, including a senatorship which was bestowed by royal favor in 1904. He was simple, almost austere in his tastes, of rigid moral fiber, a meticulous and cautious operator, and a kindly and interesting teacher. It is not surprising that he enjoyed the affection as well as the admiration of his associates and pupils. His hobby was agriculture, and he spent the last years of his life in quiet retirement, tending his garden. He died in Verona, at the age of 80, on July 19, 1924.

Bassini's contribution to the surgery of hernia was so immediately recognized to be of prime importance that it is surprising to find, in almost all discussions of the subject, confusing and contradictory accounts as to the place, time, and manner of his original publications. As Halsted tells it, Bassini first published a description of his operation in a brochure, "which anticipated my first report by at least a month or two." Halsted says, "the precise date of that pamphlet's appearance is not given," and it may, or may not, have anteceded his first operation, "in any event, I had not heard of Bassini's operation until the German article appeared—possibly about one year after my first operation; neither was I, nor any American, or German, as far as I know, aware of Bassini's first report, until the appearance of the second."

Bassini's first announcement of his operation was before the Italian Society of Surgery on April 7, 1887, in Genoa, in a paper entitled "Sulla Cura Radicale dell'Ernia Inguinale." In this presentation, Bassini reported upon 42 herniorrhaphies in 38 patients, 22 of whom had been followed for from 6 to 28 months without recurrence, the others for less than 6 months.

Bassini's second announcement of his operation entitled, "Nuovo Metodo per la Cura Radicale dell'Ernia Inguinale" was made before the Italian Medical Association at the Twelfth Congress in Pavia, in September, 1887. His presentation is reported in the Proceedings of the Congress. It is stated that he had performed 72 operations for the radical cure of inguinal hernia on 64 patients, 63 males and 1 female. The patients were discharged from the hospital in 10 to 25 days; 42 of the patients were re-examined after 6 to 31 months, and 25 patients at 1 to 5 months after operation. Five patients were lost to follow-up after recovery. The author presented a testimonial letter from a Medical

Officer attached to the Fourth Artillery Regiment, stating that, after operation, he had been able to return to full duty, and that he was perfectly healed. The following year, at the meeting of the Italian Surgical Society in Naples, Bassini presented 100 cases operated upon by his method. He was now actually reporting upon 102 hernias in 92 patients, and said that all 92 patients recovered satisfactorily, without accidents or complications. None of the patients wore trusses after operation. In 72 cases the cure was confirmed from 6 to 37 months after operation, and in 30 cases, from 1 to 5 months. He then presented, once more, the testimonial from the Captain of Artillery.

None of these three brief reports is illustrated, and, in each, the operation itself is only very briefly discussed. The papers concern themselves with an analysis of the kinds of hernias treated, and the results. In 1889 there appeared the little 106 page book to which Halsted referred, entitled "Nuovo Metodo per la Cura Radicale dell'Ernia Inguinale" published in Padua by the House of Prosperini. The manuscript is dated by the author as "Padova 30th July, 1889." The operation is described in detail and there is a tabular history of the case reports of all of his patients. The beautiful color illustrations of the Bassini operation reproduced here are taken from a copy of this book which is in the Monuments of Medicine collection in the National Medical Library. These illustrations and the text of the book, without the case reports, have been reprinted in the 1937 Fiftieth Anniversary volumes in honor of Bassini and his operation, which were published by the University of Padua.

The publication which made Bassini's work known outside of Italy, and almost at once in this country, as well as in Europe, was his paper entitled "Ueber die Behandlung des Leistenbruches" in the *Archiv für klinische Chirurgie,* in 1890. The author's date on this paper, as on the Italian book, is July 30, 1889. The copy of this journal, to which we have access, is that from Dr. Halsted's own library and now in the Welch Medical Library. Numerous items are underlined in pencil, and there are one or two marginal pencilled notes in what appears to be Halsted's handwriting.

Comparison of the Italian pamphlet with the German journal shows the *Archiv für klinische Chirurgie* paper to be a verbatim, line-for-line translation of the Prosperini pamphlet. The larger pages and smaller type-face of the German publication, together with the listing of the case reports, which had originally been published in tabular form, effect the reduction from 106 pages to 47.

In these publications Bassini stated that he became interested in the treatment of inguinal hernia in 1883, and that in the last 6 years he had studied or operated upon 274 hernias. After trying the operations of Wood and Czerny without satisfaction, in the beginning of 1883 he began to modify the operative attack upon inguinal hernia, in order to produce a radical cure, such that the patient would not require the use of a truss after operation. "Seven times I operated in the following fashion: After opening the anterior wall of the inguinal canal where the hernia presented, I isolated the neck of the sac at the internal inguinal ring. Here I applied the running suture, resected the remainder of the sac, and let the neck retract." (p. 433.) The neck of the sac

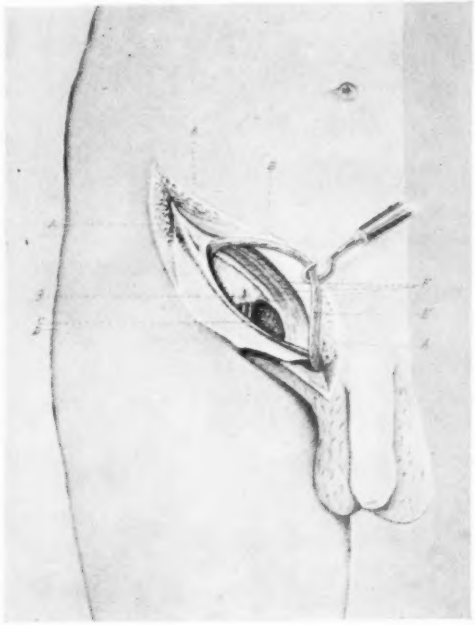


Fig. 1.

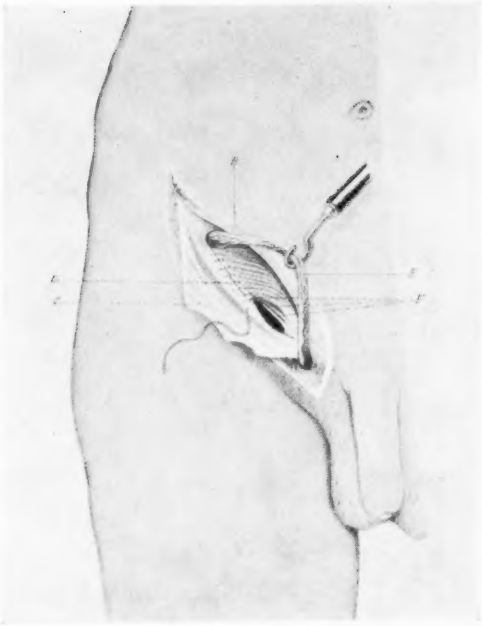


Fig. 2.

Fig. 1. *a*, The subcutaneous fat. *b*, The upper aponeurotic flap. *c*, The lower aponeurotic flap. *d*, The posterior rim of Poupart's ligament. *e*, The spermatic cord. *f*, The conjoined tendon, internal oblique muscle, transverse muscle, and the vertical fascia of Cooper. *g*, The transversalis fascia.

The aponeurosis has been split and the two aponeurotic flaps dissected back. The spermatic cord has been isolated and delivered. The neck of the hernial sac has been isolated down to the very mouth of the sac, sutured, and the excess sac excised. The amputated neck of the sac has retracted. (From Bassini: Nuovo Metodo per la Cura Radicale dell'Ernia Inguinale, Padua, 1889. Reproduced courtesy of Ethicon, Inc.)

Fig. 2. The isolated spermatic cord is lifted up and dissection continued until the developed conjoined tendon can be approximated without difficulty to the posterior edge of Poupart's ligament. These two parts are now sutured together for a length of 5 to 7 cm. from the pubis toward the spermatic cord, which is displaced backward about 1 cm. from the anterior superior spine of the ilium. The closure is a continuous one. Note that Bassini placed no sutures above or lateral to the emergence of the cord. (From Bassini: Nuovo Metodo per la Cura Radicale dell'Ernia Inguinale, Padua, 1889. Reproduced courtesy of Ethicon, Inc.)

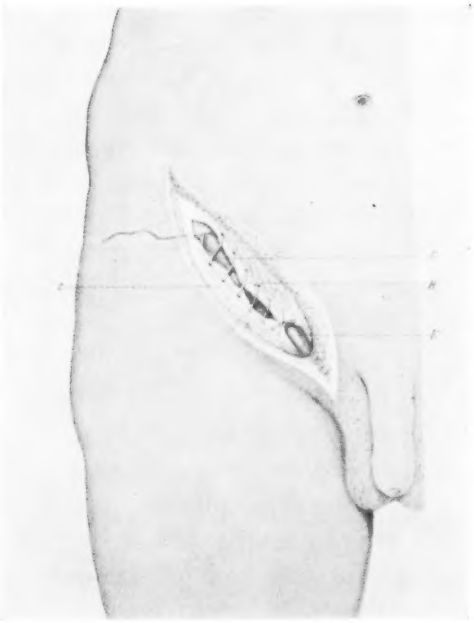


Fig. 3.

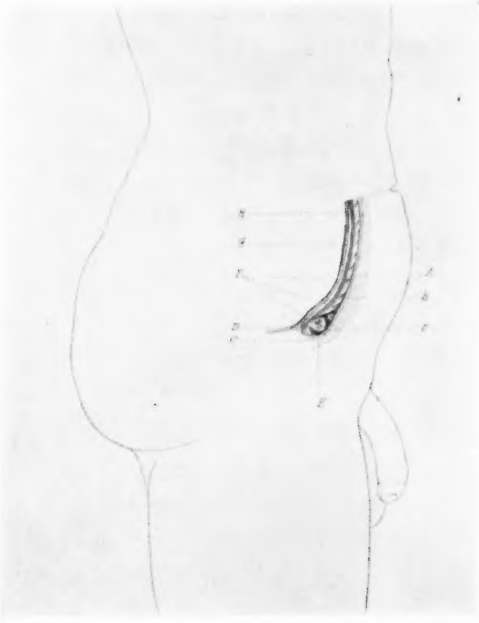


Fig. 4.

Fig. 3. The spermatic cord has been replaced and the flaps of the external oblique aponeurosis sutured. (From Bassini: Nuovo Metodo per la Cura Radicale dell'Ernia Inguinale, Padua, 1889. Reproduced courtesy of Ethicon, Inc.)

Fig. 4. The diagram shows schematically the situation after the repair has been completed. The conjoined tendon *f* has been sutured to the posterior rim of Poupart's ligament *d* behind the cord *c*. The aponeurotic flaps (*b* and *c*) have been joined together, anterior to the cord, and between it and the skin. (From Bassini: Novo Metodo per la Cura Radicale dell'Ernia Inguinale, Padua, 1889. Reproduced courtesy of Ethicon, Inc.)

was sutured to the posterior surface of the aponeurosis of the external oblique in the region of the abdominal ring, and a tampon constructed of the projecting portion of the neck of the sac.

He performed 7 such operations in a relatively short time. One of these patients died 3 months later, of a cause unrelated to the hernia and the operation for it. Autopsy showed the sutured portion of the neck of the sac completely resorbed, so that Bassini felt that such patients, although rendered more comfortable, still would have to wear trusses to prevent recurrence. Continuing to seek a method for the radical cure of hernia, "I thought that this might well be achieved through transplantation of the inguinal canal as in the physiological condition, that is to say, a canal with two openings, one abdominal and one subcutaneous, and, furthermore, with two walls, one anterior and one posterior, through the middle of which the spermatic cord would pass. Through a study of the groin, with the help of an anatomical knowledge of the inguinal canal and inguinal hernia, it was easy for me to find an operative method which answered the above described requirements, and made possible a radical cure without requiring the subsequent wearing of a truss." (p. 435.) He further stated that he had used this method exclusively since the year 1884, and had operated in 227 patients upon 262 hernias, of which 11 were strangulated and some of the remaining 251 irreducible. He described the operation as follows (pp. 436-438):

> 1. In one incision the skin is incised in the inguinal scrotal region, the aponeurosis of the external oblique laid bare, and the subcutaneous inguinal ring exposed.
>
> 2. The aponeurosis of the external oblique is incised from the external inguinal ring to the region of the internal ring, then the aponeurosis dissected above and below in two flaps, the spermatic cord isolated and delivered, as well as the neck of the hernial sac. The neck of the hernial sac is isolated down to the mouth of the hernia and separated from the elements of the spermatic cord. The isolation of the neck of the sac must be continued down to the very mouth of the sac; the body and fundus of the sac are then isolated and opened and the contents are examined. The thickened omentum is removed and the contents of the sac replaced. A ligature is placed about the neck of the sac while tension is made on it, and the sac cut 0.5 cm. distal to the ligature. When the neck of the sac is very wide, additional sutures are placed through the neck to ensure closure of the sac. The amputated neck of the sac retracts.
>
> 3. The isolated spermatic cord is easily lifted up out of the abdominal wall, with the testis, if necessary, which is drawn up out of the scrotum. The upper flap of the aponeurosis of the external oblique is drawn upward, and the lower flap down; the latter is dissected down to Poupart's ligament, exposing the internal ring 1 cm. above the exit of the spermatic cord. Medially the anterior sheath of the rectus abdominis is freed, and the conjoined tendon, composed of the internal oblique, the transversus, and Cooper's vertical fascia, is separated from

the aponeurosis of the external oblique and the subcutaneous adipose tissue. This dissection is continued until the developed conjoined tendon can be approximated without difficulty to the posterior isolated rim of Poupart's ligament. These two parts are now sutured together for a length of 5 to 7 cm. from the pubis toward the spermatic cord, which is displaced backward about a centimeter from the anterior superior spine of the ilium.

Halsted, if, as we think, the pencilled notations in the margins are his, bracketed the following sentence: "The first two sutures barely in the pubis also take in the anterior sheath of the rectus abdominis."

Bassini went on to state that, if at this point in the operation the patient is stimulated to vomit, which he had attempted in his first 50 cases, the inguinal region would sustain the greatest intra-abdominal pressure ever exerted, and that the conjoined tendon fixed to Poupart's ligament was "sewed tightly and remains immobile in its new place."

4. The spermatic cord is now replaced, and the testis as well, by suture of the aponeurosis of the external oblique and then the skin. He drained only voluminous hernias of long standing requiring extensive dissection. In this manner he states the inguinal canal is reconstructed with an inner opening and a posterior wall, both made of the conjoined tendon fixed to the posterior edge of Poupart's ligament and an anterior canal wall, composed of the joined flaps of external oblique aponeurosis, with a narrow external or subcutaneous opening.

Bassini stated that 11 of the 262 hernias were strangulated, although many others were irreducible. In 35 cases, simultaneous bilateral operations were performed. The patients varied in age from 13 months to 69 years. From a careful reading of the individual case reports we find 3 deaths, and these all in strangulated hernia cases—No. 69, dead in 22 days of pyemia, No. 78, dead in one day of shock, and No. 201, dead in 15 days of pneumonia which had its onset on the ninth day. There were 11 instances of infection. We find a tabulation of 8 recurrences in 6 patients. In making his own count of recurrences, Bassini overlooked Case 65. The operations in the case reports are dated from December, 1884, to June, 1889, and the follow-up examinations were principally in June and July of 1889. The longest follow-up, in the first case, was 4½ years.

REFERENCES

Bassini, Edoardo: Sulla Cura Radicale dell'Ernia Inguinale, Archiv. Soc. Ital. d. Chir. 4: 380, 1887.
(We have not been able to find this volume in any of the medical libraries in this country. However, Gustavo Lusena, in a book entitled *La Societa Italiana di Chirurgia Nei Suoi 30 Congressi (1883-1923)*, Roma, 1934, Manuzio, on pages 284 and 285 published the complete summary of Bassini's remarks.)

Bassini, Edoardo: Nuovo Metodo per la Cura Radicale dell'Ernia Inguinale, Atti. d. Cong. d. Ass. Med. Ital. 2: 179-182, 1887.

Bassini, Edoardo: Sopra 100 Casi di Cura Radicale dell'Ernia Inguinale Operata col Metodo dell-autore, Archiv. ed. atti. d. Soc. Ital. di Chir. 5: 315-319, 1888.

Bassini, Edoardo: Nuovo Metodo per la Cura Radicale dell'Ernia Inguinale, Padua, 1889, Prosperini.

Bassini, Edoardo: Ueber de Behandlung des Leistenbruches, Arch. f. klin. Chir. 40: 429-476, 1890.

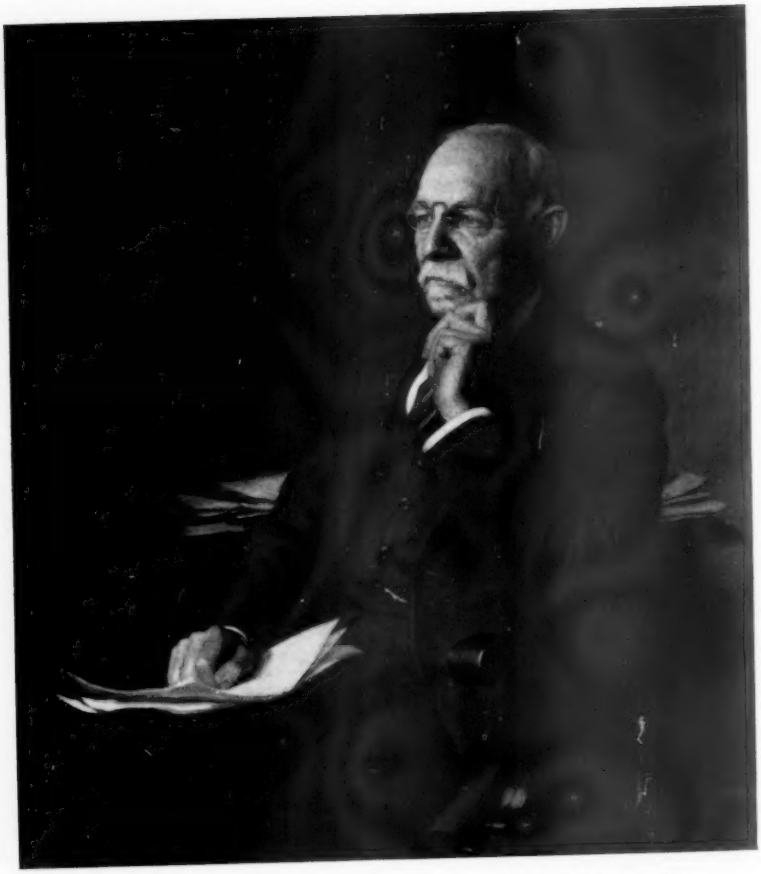
WILLIAM STEWART HALSTED
1852—1922

Part I

Halsted was born in New York City. His undergraduate training was at Yale University, where he was chiefly distinguished for his athletic prowess. He attended Medical School at the College of Physicians and Surgeons, in New York, and during this time interned at Bellevue Hospital. After graduation he served as a house officer at the New York Hospital. He then went to Europe for 2 years of study in Vienna and elsewhere. He came to know Billroth and Mikulicz, von Eiselsberg, Kocher, and others, with whom he maintained a lifelong association, refreshed frequently on long European trips.

Returning to New York, Halsted was on the staff of several hospitals, including the Roosevelt and Bellevue. He vigorously sponsored the introduction of the new antiseptic techniques into the hospitals of the city, organized the Outpatient Department at the Roosevelt Hospital, maintained an extremely busy and successful surgical practice, and, in the custom of the day, taught a "quiz" course for medical students, which was said to be the most popular and stimulating in the city. He wrote on fractures of the hip, on blood transfusion (transfusing his own blood into his sister when she was dying of a postpartum hemorrhage) and successfully performed a cholecystostomy on his own mother, who had been given up by the consultant who had seen her.

During this period he conducted his famous experiments on the possible uses of cocaine for the production of local anesthesia, at a time when its habit-forming properties were unknown. Addiction followed, and it is notable that his co-workers who became similarly addicted all eventually succumbed to its effects. Halsted made a strenuous effort to break himself of the addiction and returned to a long, fruitful, and vigorous professional life. No evidence has been adduced to indicate that he was not completely cured. As Dr. E. A. Park pointed out at the time of the Halsted Centennial, it really made very little difference. If he had completely cured himself of the habit, it was a remarkable feat of will power, and if he had not completely cured himself, his extraordinary career is all the more credit to one laboring under this handicap. Called to Hopkins in 1886, he founded the first true University Department of Surgery in this country, and the present residency systems the country over are a direct consequence of his modification of the German system of postgraduate training. He was a patrician of the intellect, remote, austere, precise.



WILLIAM STEWART HALSTED

Halsted's major works were certainly those on carcinoma of the breast, on inguinal hernia, and on goiter. He made numerous technical contributions of which the use of rubber gloves is the best known. More important is his influence on American surgery, through his residents, who filled numerous chairs of surgery, and continued his emphasis on the prime surgical principles of precise hemostasis, asepsis, and avoidance of tissue trauma, and on the use of experimental animals and the laboratory in the investigation of surgical problems. His writings are masterpieces of lucidity and precision in expression.

Halsted's first communication on the subject of hernia was made before The Johns Hopkins Hospital Medical Society in Baltimore on Nov. 4, 1889. The proceedings were reported in *The Johns Hopkins Hospital Bulletin,* in 1890. The paper was entitled "The Radical Cure of Hernia." The report stated that "Dr. William S. Halsted presented five patients upon whom he had performed his operation for the cure of inguinal hernia. He described the operation as follows [p. 261*]:

> "1. The incision begins at the external abdominal ring, and ends one inch or less (less than one inch in children) to the inner side of the anterior superior spine of the ilium on an imaginary line connecting the anterior superior spines of the ilia. Throughout the entire length of the incision *everything superficial to the peritonaeum is cut through.*
>
> "2. The vas deferens, with its vessels, is carefully isolated up to the outer termination of the incision, and held aside.
>
> "3. The sac is opened and dissected from the tissues which envelop it.
>
> "4. The abdominal cavity is closed by quilted sutures passed through the peritonaeum at a level higher by 1½-2 inches than that of the so-called neck of the sac.
>
> "5. The vas deferens and its vessels are transplanted to the upper outer angle of the wound.
>
> "6. Interrupted, strong silk sutures, *passed so as to include everything between the skin and the peritonaeum,* are used to close the deeper portion of the wound, which is sewed from the crest of the pubis to the upper outer angle of the incision. The cord now lies superficial to these sutures, and emerges through the abdominal muscles about one inch to the inner side of the anterior superior spine of the ilium.
>
> "7. The skin is united over the cord by interrupted stitches of very fine silk. These stitches do not penetrate the skin, and when tied they become buried. They are taken from the underside of the skin, and made to include only its deep layers—the layers which are not occupied by sebaceous follicles."

It is clear from step No. 6 that the entire repair of the defect was made with one layer of rather heavy sutures. The report suggests further that Dr.

*Page references to Dr. Halsted's papers are to Volume I, Surgical Papers, by William Stewart Halsted, Baltimore, 1924, The Johns Hopkins Press.

Halsted was as much interested in the subcuticular closure of the skin as in the actual hernia repair. The report continues:

> "8. One or two small, short gauze plugs are used as wound drains." The after treatment: "The gauze plugs are removed at the first subsequent dressing—usually at about the 7th day. The patients are allowed to walk about on the 21st day."

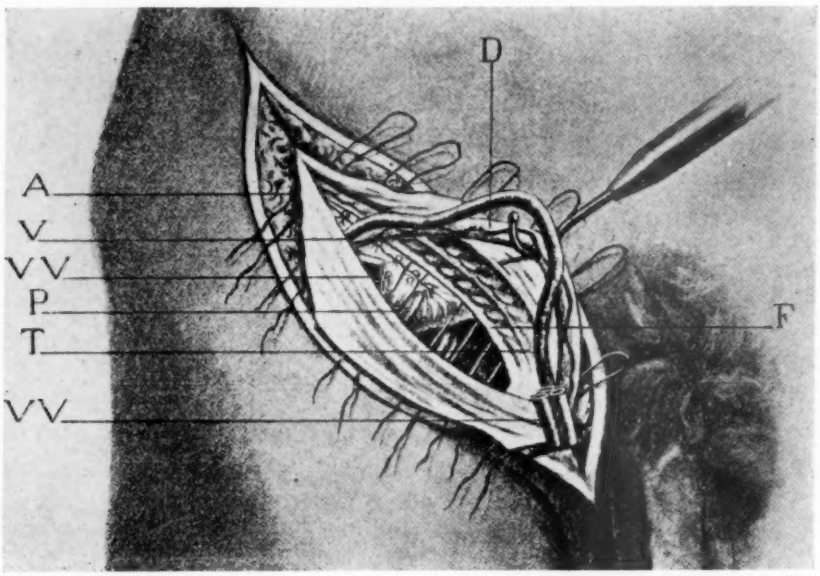


Fig. 5. The first step of the operation. Indicated is the skin incision which "extends from a point about five centimeters above, and external to the internal abdominal ring to the spine of the pubes." (p. 273.) The aponeurosis of the external oblique muscle, the internal oblique and transversalis muscles, and the transversalis fascia are "cut through from the external abdominal ring to a point about 2 cm. above and external to the internal abdominal ring." Halsted laid stress, at this time, on dividing the internal oblique from the inguinal ligament, thus moving the internal ring laterally and superiorly. The excess veins of the cord have been excised, and the cord is held out of the way with a blunt hook. The single row of sutures completing the entire repair has been placed, but not tied. The sac which has been isolated has been closed in a linear fashion high above its "neck" and the excess excised. The sutures consist of "six or eight deep mattress sutures which pass through the aponeurosis of the external oblique and through the internal oblique and transversalis muscles and transversalis fascia on the one side and through the transversalis fascia and Poupart's ligament and fibers of the aponeurosis of the external oblique muscle on the other."

The 5 cases were briefly summarized. All of the patients recovered uneventfully except a 48-year-old man, described as a German and a blacksmith, who, 10 days after operation, "passes urine through wound." It was inferred that one of the deep sutures was passed through the wall of the bladder.

September 4, "Patient passed all of his urine through the penis."

September 17, "Patient is out of bed. The wound is healed except at its lower angle."

Note that, at this stage, in addition to the relative crudeness of the repair, Halsted used drains, something he later took pride in avoiding.

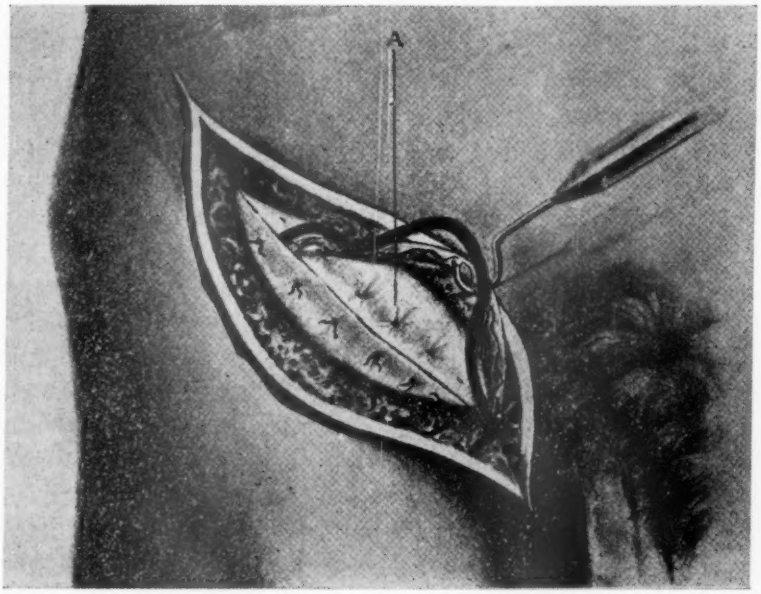


Fig. 6. Second step—the sutures tied. Halsted pointed out that the point of emergence of the cord at the upper angle is determined by the condition of the tissues. If the muscles at the internal abdominal ring are thick and firm, the cord is brought out there. If they present thin edges, the cord is transplanted farther out. The sutures above and below the cord, he considers "the most important stitches, for the transplanted cord passes out between them." It will be seen that there is no imbrication or overlapping of the tissues, and that there is no more than a single layer of mattress sutures.

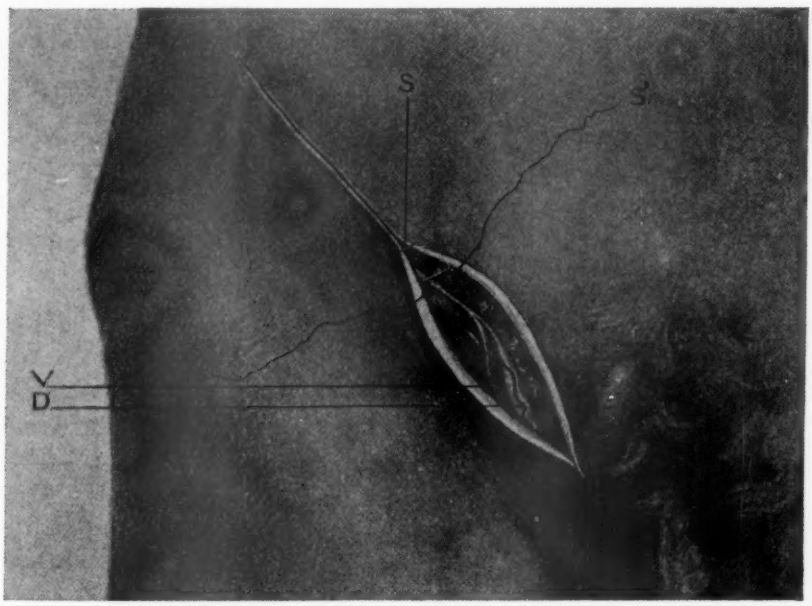


Fig. 7. Third step—a buried interrupted subcuticular suture of fine silk. Halsted laid great stress on this as avoiding contamination from the skin. He said nothing about the use of sutures in the subcutaneous tissues.

Almost exactly 12 months later at The Johns Hopkins Medical Society, on Oct. 20, 1890, Halsted made a further report which was published the same year in *The Johns Hopkins Hospital Bulletin.* The occasion was the presentation of a patient with an interesting complication, thrombosis of the femoral vein. Halsted stated, "We take such deep stitches into the pillars of the ring that I am not surprised at this complication. In one case we passed a stitch through the wall of the bladder." He added that in the 12 patients he had now operated upon there had been, thus far, no recurrences.

Again before The Johns Hopkins Medical Society in January, 1892, Halsted spoke on hernia to discuss the excision of most of the veins of the cord.

His first extensive paper, "The Radical Cure of Inguinal Hernia in the Male," was read at the annual meeting of the Medical and Chirurgical Faculty of Maryland in Easton, on Nov. 17, 1892, and published in both *The Johns Hopkins Hospital Bulletin* and the *Annals of Surgery* in 1893. After discussing the lamentable state of the surgery of inguinal hernia at that date, and commenting at length on Bull's analysis of his own results with only 4 patients free from recurrence out of 136 observed for over 4 years, Halsted indicated that this was the general state of the operation, and that "the majority of surgeons operate for the radical cure of hernia only when the hernia is strangulated or cannot be retained with a truss." Halsted pointed out that, aside from some advance in instruments, the technique of herniotomy at the time was "precisely that of Pierre Franco, the gifted stone and rupture cutter, of the 16 Century." His historical study of the development of the cure of inguinal hernia up to that time is authoritative, detailed, and fascinating.

As to the timing of his own work, Halsted states, "More than three years ago I described a new operation for the cure of inguinal hernia in the male. Six or eight months later, Bassini, of Padua, published his operation for the cure of inguinal hernia, which he had performed 251 times, with only seven returns, and no deaths, except one, and that from pneumonia after the wound had healed. Bassini's operation and mine are so nearly identical that I might quote his results in support of my operation." (p. 273.)

In this paper, for the first time, Halsted gave a detailed description of his operation (p. 273):

> "Instead of trying to repair the old canal and the internal abdominal ring, as McEwen had tried to do, I make a new canal and a new ring. The new ring should fit the cord as snugly as possible, and the cord should be as small as possible. The skin incision extends from a point about five centimeters above and external to the internal abdominal ring to the spine of the pubis. The subcutaneous tissues are divided so as to expose clearly the aponeurosis of the external oblique muscle and the external abdominal ring. The aponeurosis of the external oblique muscle, the internal oblique and transversalis muscles and the transversalis fascia are cut through from the external abdominal ring to a point about 2 cm. above and external to the internal abdominal ring. The vas deferens and the blood vessels of the cord

are isolated. All but one or two of the veins of the cord are excised.... The sac having been completely isolated and its contents replaced, the peritoneal cavity is closed by a few fine silk mattress sutures, sometimes by a continuous suture. The sac is cut away close to the sutures. The cord, in its reduced form, is raised on a hook out of the wound to facilitate the introduction of the six or eight deep mattress sutures, which pass through the aponeurosis of the external oblique and through the internal oblique and transversalis muscles and transversalis fascia on the one side, and through the transversalis fascia and Poupart's ligament and fibres of the aponeurosis of the external oblique muscle on the other.

"The two outermost of these deep mattress sutures pass through muscular tissues, and the same tissues on both sides of the wound. They are the most important stitches, for the transplanted cord passes out between them. If placed too close together, the circulation of the cord might be imperiled, and if too far apart, the hernia might recur."

There is no question then but that at this time, after a high ligation of the sac, Halsted transfixed all of the layers of the abdominal wall medial to the sac and brought them over to the lateral structures, carrying the suture out through the transversalis fascia, transversalis muscle, inguinal ligament, and aponeurosis, all in one bite, and tied this single layer of sutures down to secure his repair. The cord in this operation was left subcutaneous. He pointed out that his operation differed to some extent from Bassini's (p. 274):

"1. Bassini always brings the cord through the muscles at the internal abdominal ring. The point out to which I transplant the cord is determined, as I have said, by the condition of the muscles.

"2. Bassini does not excise the superfluous veins. I believe that it is advisable to reduce the size of the cord as much as is practicable.

"3. In Bassini's operation the cord lies posterior to the aponeurosis of the external oblique muscle; in mine between this aponeurosis and the skin."

He makes the rather trivial point that Bassini requires an additional row of sutures to secure the cord in that position, and that he thinks this row of sutures might be spared, unless it should be shown that there was something to be gained by them.

Discussing the time the patient should stay in bed, he stated, "our patients are kept upon their backs for 21 days," and went on, "I sometimes question the propriety of allowing, as I do, my patients to walk about on the 21st day." He then spoke in general about the surgical technique, and stated: "It is not always possible to bring together the pillars of the external abdominal ring without a little tension. One can, of course, make relaxation cuts, but these would be quite as undesirable as a moderate amount of tension." Quite apparently at this time the relaxation incision had not yet come into use. He had also given up the use of gauze drains, for he stated: "We never resort to

drainage of any kind for fresh wounds." He was able to assert that since the opening of The Johns Hopkins Hospital 3½ years before, 82 operations for the radical cure of hernia had been performed without a death. In 58 of the cases the operation detailed above had been employed, and he added, "Of the cases which healed per primam, not one has recurred. The wounds which suppurated were immediately laid wide open and allowed to heal by granulation. For the result in such cases the open method, and not mine, is responsible." He stated that in these there had been 6 recurrences. It is interesting that Halsted was not above blaming patients for his failures, for he said, "No. 2 took cathartics and got out of bed a few days after the operation. He was discharged for insubordination on the eighth day, before his wound was firm." The paper reports individually every one of the 82 herniorrhaphies that had been done in the previous 3½ years.

At a meeting of The Johns Hopkins Medical Society on May 7, 1894, in a paper published the same year in *The Johns Hopkins Hospital Bulletin,* Dr. Halsted presented a "Report of Twelve Cases of Complete Radical Cure of Hernia by Halsted's Method of Over Two Years' Standing. Silver Wire Sutures."

He presented a patient who had been operated upon only 2 weeks before, in whom both the deep buried sutures and the continuous buried skin sutures were of silver wire. He pointed out that experiments had shown that after zinc, cadmium, and copper, silver was the most effective metal in inhibiting the growth of organisms, and added that, since the report of the year before, they had had "a great many cases of hernia" with no known recurrences. In the 12 cases presented there were no recurrences.

In 1895, in the *American Journal of the Medical Sciences,* Halsted published a brief paper describing his thoughts concerning "The Operative Treatment of Hernia." He was still enthusiastic about the reduction of the size of the cord by excising the veins when they formed a large bundle, but was forced to add in a footnote that he had finally seen 3 cases of atrophy of the testicle, presumably as a result of excision of the veins. In this paper he indicated that at times the cremaster muscle was included in the single layer of sutures repairing the hernia. He stated that they had now operated 165 times for the cure of various forms of hernia in both sexes without a death. One hundred and six males with inguinal hernia had been operated upon by his method. Thus far there still had been no recurrences in patients whose wounds had healed per primam.

He stated rather testily (p. 289), "I dislike to have my operation referred to as a modification of Bassini's operation. The operations are undoubtedly original with both of us, and mine was described several months before we had heard of Bassini's operation." He went on to point out that in his operation the cord was transplanted out into the thicker muscle and was brought superficial to the aponeurosis of the external oblique. He thought that in Bassini's operation the suturing of the aponeurosis near Poupart's ligament would result in impairment of the circulation of the aponeurosis, and criticized Bassini's claim

that his operation re-established the obliquity of the canal. "The original canal is not by any means an affair so simple as Bassini's. To reproduce the equivalent, anatomically and physiologically, of the inguinal canal, is, for us, impossible." He stated that for about a year he had used silver sutures in all of his hernia operations, and had covered all of the wounds with silver foil. He had tried copper and brass foil, and copper and brass wires previously, but the corrosion of the tissues caused him to cease.

REFERENCES

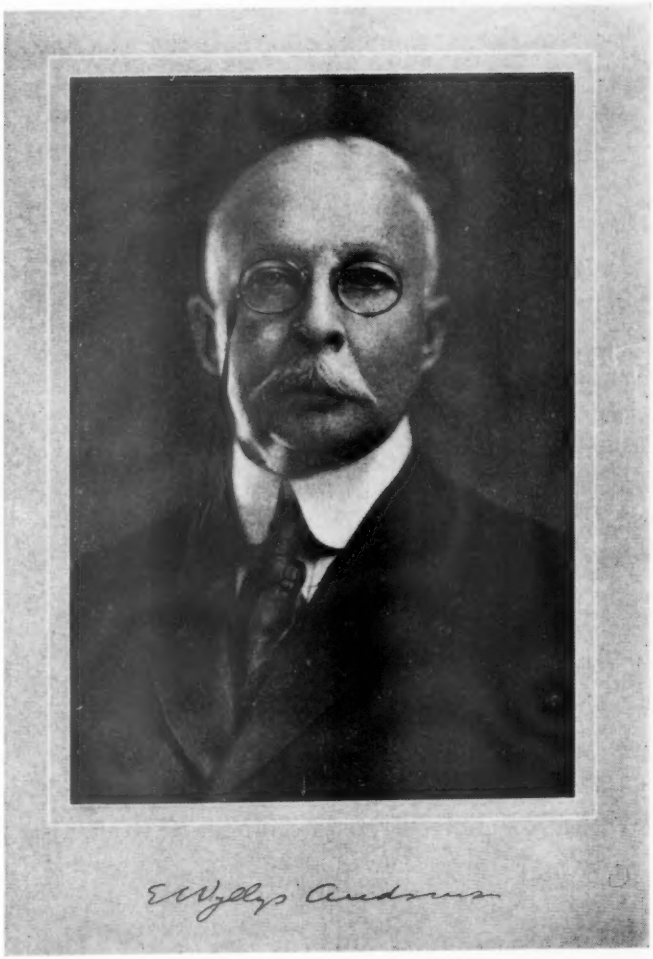
Halsted, W. S.: The Radical Cure of Hernia, Johns Hopkins Hosp. Bull. **i**: 12-13, 1890.
Halsted, W. S.: The Radical Cure of Hernia, Johns Hopkins Hosp. Bull. **i**: 112, 1890.
Halsted, W. S.: Excision of Some of the Veins of the Cord in the Operation for the Radical Cure of Inguinal Hernia, Johns Hopkins Hosp. Bull. **iii**: 76, 1892.
Halsted, W. S.: The Radical Cure of Inguinal Hernia in the Male, Johns Hopkins Hosp. Bull. **iv**: 17-24, 1893; Ann. Surg. **xvii**: 542-556, 1893.
Halsted, W. S.: Report of Twelve Cases of Complete Radical Cure of Hernia by Halsted's Method of Over Two Years' Standing. Silver Wire Sutures, Johns Hopkins Hosp. Bull. **v**: 98-99, 1894.
Halsted, W. S.: The Operative Treatment of Hernia, Am. J. M. Sc. **110**: 13-17, 1895.
Halsted, W. S.: Discussion on the Operative Treatment of Hernia, Tr. Am. S. A. **xiii**: 343-344, 1895.

E. WYLLYS ANDREWS
1856—1927

Andrews, at the height of his career, was Professor of Surgery at the Northwestern University Medical School and had been Surgeon-in-Chief at the Cook County Hospital, and Attending Surgeon at the Michael Reese and St. Luke's Hospitals in Chicago. He was one of the founders of *Surgery, Gynecology and Obstetrics,* and one of the founding members of the Association for Thoracic Surgery. The testimony of his colleagues is always to a remarkable evenness and kindness of person, profoundness of scholarliness, and beauty of surgical technique. In a city, and at a time, when surgeons were colorful and their relations impetuous, he had "a total lack of all quarrelsomeness and opposition" and "never have I known him to say an unkind word or thing of one of his associates, no matter what the provocation. If he could not praise, he said nothing." He was thought to be a competent Latin scholar and a real student of Shakespeare and his library was rich in Shakespeariana. "His technical surgery was a wonderful display of efficiency and action, for he never made an unnecessary or useless movement. While he seemed deliberate, so well did he control each purposeful action, that his surgery was unusually rapid. His delicacy, his knowledge of anatomy, and his gentleness in all thought and action, brought him constant successful results." His paper on hernia is beautifully written.

E. Wyllys Andrews, of Chicago, a meticulous surgeon, in 1895 contributed the principle of imbrication of the flaps in hernia repairs: "While based upon the most modern, open method, and while confessedly an outgrowth of experience with the McEwen, Bassini, Halsted and similar operations, yet the carrying out of the imbrication idea so far changes the technique as to make it as different from them all as they are from each other, and perhaps entitles it to the term 'A new operation.'" He stated: "Every surgeon who has followed the subject through the evolution of the last six years must rejoice that the recent progress has been so rapid. Probably most operators used instinctively to feel, as I did, that they were groping in the dark, and had evolved in their own minds the very step that Bassini has invented. Dr. Marcy goes further and asserts that his own operation, as described some years ago, is practically the same as that called the Bassini." Andrews said of previous operations for hernia that "of all surgical procedures, this one seems to have suffered most from useless timidity, blind conservatism, and perverted ingenuity."

He had some remarks to make on the general technique of surgery. "Let me say that an extended experience with drainage, for a long time, for a short time, and with hermetically sealed undrained wounds, has led me to some very positive conclusions on this feature. I assert most emphatically that the sealed aseptic wound will remain aseptic in this locality and that no other will."



Halsted employed gauze drains in his earlier operations and had recorded his abandonment of drains in his 1892 paper three years prior to the appearance of this paper of Andrews'. Andrews lays great stress on an elaborate preparation of the surgeon's hands and of the skin of the abdomen which is prepared for 24 hours before operation. "I use only sterile water irrigation (of the wound) or none at all, believing that bichloride solutions cause superficial cell necrosis unfavorable to the quickest union and even promoting sepsis by furnishing a medium. Finally, I unite the skin itself by a buried suture which does not puncture any of its glands or ducts. There is no after-dressing to such wounds, save, possibly, the occasional re-application of the collodion if it becomes loosened." It is interesting to see the evidence of such sound surgical thinking, respect for tissues, and meticulous technique, obviously arising independently in Chicago at the same time that Halsted was stressing many of the same points in Baltimore.

Andrews' technique may be described as follows:

1. The incision is parallel to Poupart's ligament and from the pubis to a point above the internal ring. He continued to emphasize careful technique: "All steps of this operation must be clean and bloodless. If hemostosis [sic] is delayed to the close of the operation it will be simply impossible to carry on the deep dissection and suturing carefully and the final sealing with collodion will probably be followed by more or less hematoma, necessitating after drainage, defeating deep primary union, and even favoring suppuration. To bury the sutures successfully they must be in contact with living tissue cells, not entangled in coagula."

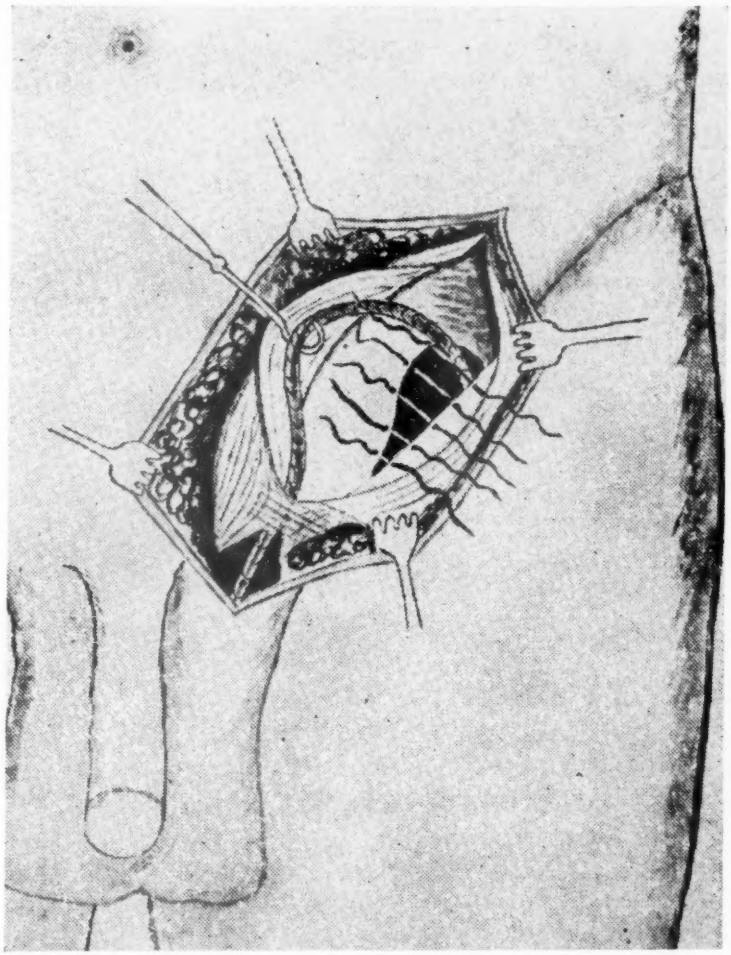
2. An incision of the same length and direction as the skin incision is made through the external oblique, terminating near the external ring. He found it not always necessary to divide the ring.

3. The sac is dissected out, cross sutured at its base, cut short, and returned to the peritoneal cavity. "I attach no value whatever to the McEwen plan of forming it into a plug, or to Kocher's twisting method."

4. The cord is gently lifted from its bed. "We now come to that step of Bassini's method which is most original and important. The posterior wall of the canal up to and including the internal ring is narrowed by suturing the conjoined tendon and transversalis fascia firmly to Poupart's ligament." He used Marcy's kangaroo tendon. "So closely should the line of sewing be carried to the inner ring that its aperture is narrowed until it barely admits the cord. I do not advocate the removal of all except the vas deferens and one or two vessels in order to reduce the size of the cord as practiced by Dr. Halsted, of Baltimore. Nevertheless it is essential to make the ring fit around it snugly." He stated that, whereas Bassini placed the cord

above the line of the sutures and closed the aponeurosis of the external oblique over it, forming a sort of new inguinal canal, Postemski and Halsted "independently devised another and ingenious method of forming a new inguinal canal, viz.: By locating it outside the abdominal muscles altogether, making the new channel between the external oblique tendon and the skin."

5. Pointing out that the Bassini operation sometimes can be accomplished only by suturing the conjoined tendon and internal oblique to the inguinal ligament under considerable tension, and that sometimes

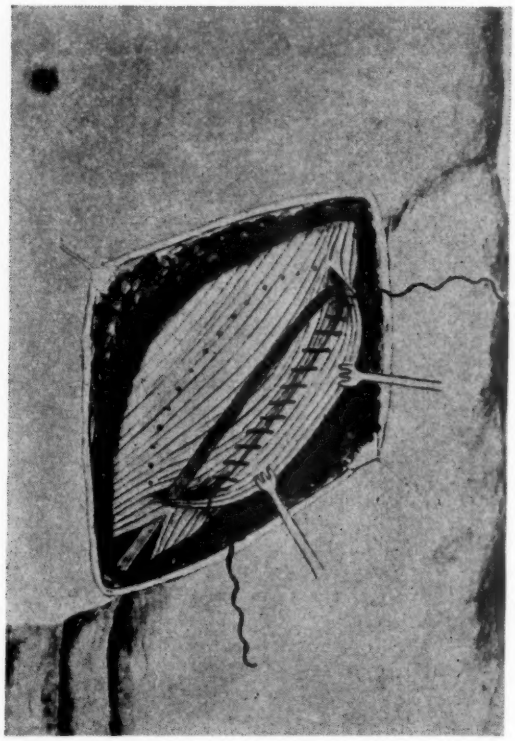


Bassini's operation, showing deep sutures ready for tying to repair internal ring.

Figs. 8 and 9 show Andrews' comparison between Bassini's operation and his own.

Fig. 8. "We now come to that step of Bassini's method which is most original and important. The posterior wall of the canal, up to, and including the internal ring, is narrowed by suturing the conjoined tendon and transversalis firmly to Poupart's ligament."

an absence of adequate conjoined tendon requires the sutures to go almost or quite to the sheath of the rectus, Andrews wrote: "the need of supplementary tissue to aid in filling this gap led me to employ the imbrication method. It consists in brief, in the incorporation of a strong layer of external oblique aponeurosis into the posterior wall of

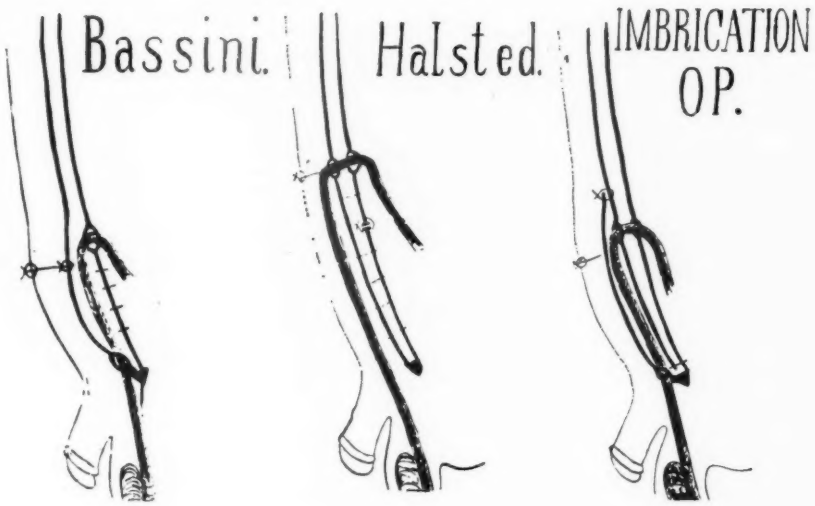


Imbrication operation showing how the deep sutures draw upper flap of external oblique down to Poupart's ligament. The lower flap shown retracted will be replaced over cord to the dotted line.

Fig. 9. "The interlocking or overlapping principle of uniting the musculo-aponeurotic layers of the abdomen is, in reality, an outgrowth of clinical experience rather than of theory." He felt that the posterior wall of the canal was weak in the Bassini repair, and that this was best strengthened by "the incorporation of a strong layer of external oblique aponeurosis into the posterior wall of the canal, along with as much conjoined tendon and transversalis fascia as can be found, and the preservation of another flap of the same strong fascia to form the anterior wall [of the canal]. The purpose is to lap joints in uniting the several layers of the wound. In the completed operation we thus have three strong layers of aponeurosis in the place formerly occupied by two, two of these being at a point of greater strain, namely, behind the cord, and all shortened or narrowed by an amount equal to the extent of overlapping. Moreover this overlapping makes the union of one surface to another exceedingly broad and firm, so that they support each other in the most admirable manner."

the canal, along with as much conjoined tendon and transversalis fascia as can be found, and the preservation of another flap of the same strong fascia to form the anterior wall. [Halsted, earlier, had included the aponeurosis in his deep repair.] The purpose is to lap joints in uniting the several layers of the wound. In the completed operation we thus have three strong layers of aponeurosis in the place formerly occupied by two, two of these being at a point of greater strain, namely, behind the cord, and all shortened or narrowed by an amount equal to the extent of overlapping. Moreover, this overlapping makes the union of one surface to another exceedingly broad and firm, so that they support each other in the most admirable manner.''

Andrews mentioned a number of possibilities in the method of imbrication. The external oblique could be overlapped and united in front of the cord ''after the manner of Lucas-Championnière,'' or the lateral aponeurotic flap could be



Schematic representation of anterior abdominal wall in antero-posterior section, showing three methods of disposal of the cord.

Fig. 10. Andrews has here indicated the difference in the position of the cord in the Bassini operation (left), the original Halsted operation with transplantation (center), and his own procedure (right).

Actually, Halsted, in his original procedure, had included the medial flap of the aponeurosis with the conjoined tendon, internal oblique, and transversalis, precisely as Andrews later did, suturing it to the inguinal ligament behind the cord. However, Halsted then brought the lateral flap of the aponeurosis also behind the cord, surely securing a stronger union of the fascial elements.

In the Bassini operation the cord is brought anterior to the union of the internal oblique with the inguinal ligament, but underneath the aponeurotic repair.

In his own imbrication operation, Andrews shows the medial aponeurotic flap being brought down with the conjoined tendon to Poupart's ligament behind the cord and the lateral aponeurotic flap sutured anterior to the cord.

used to support the posterior wall, in either case the aponeurotic flap going behind the cord, together with the internal oblique and transversalis. He drew the following:

> "Conclusions: Any successful method of radical cure must be a true plastic operation upon the musculo-aponeurotic layers of the abdominal wall. Cicatricial tissue and peritoneal exudate are of no permanent value. The following advantages are claimed for imbrication:
>
> "1. A large, strong flap of any needed size to fill the internal ring.
>
> "2. Triplicate layers of aponeurosis.
>
> "3. Interlocking of layers giving broad surfaces of union.
>
> "4. Shortening of anterior as well as posterior wall of canal, making them mutually supporting and relieving tension on deep sutures.
>
> "5. Cord amply protected."

REFERENCE

Andrews, E. W.: Imbrication or Lap Joint Method: A Plastic Operation for Hernia, Chicago M. Rec. 9: 67-77, 1895.

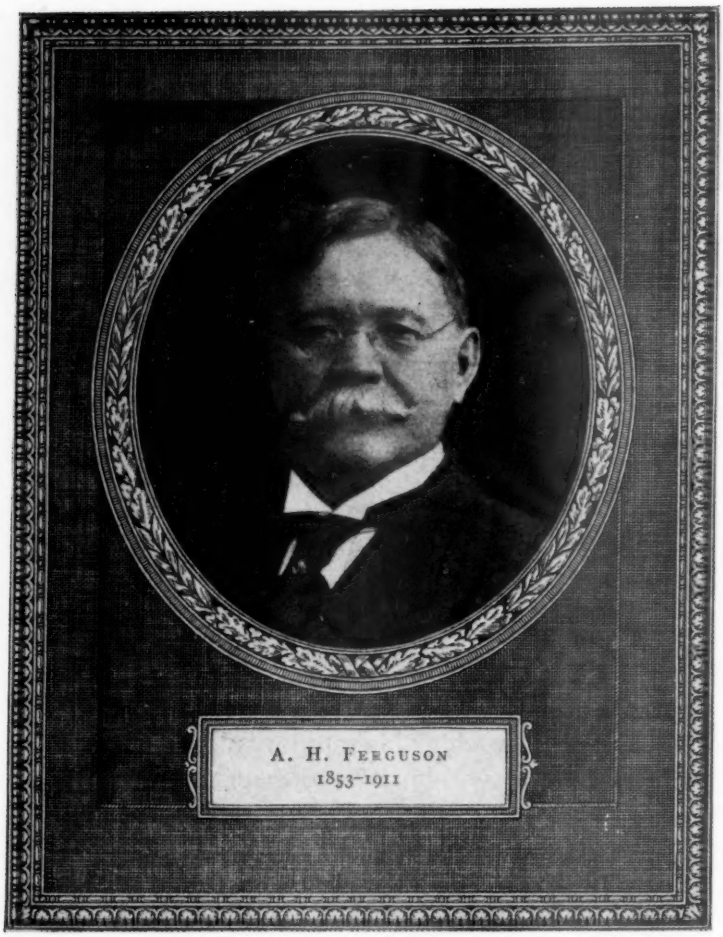
ALEXANDER HUGH FERGUSON
1853—1911

Ferguson was born in Ontario and moved early, with his family, to the remote western province of Manitoba. He was educated at Manitoba College, and received his M.B. from the Medical College of Trinity University, Toronto, and later in the same year the M.D. and C.M. He practiced surgery in Winnipeg from 1882 to 1894 and there was the prime mover in the organization of the Manitoba Medical College, in which he was first Professor of Physiology and Histology, and then Professor of Surgery. During this time he interrupted his practice for the European tour, customary for serious students of medicine in that day, and visited the centers in London, Edinburgh, Glasgow, and Berlin. For 6 months he studied bacteriology in Koch's laboratory.

By 1900 he was one of the most renowned surgeons in Canada, widely known as a clinical surgeon and as a teacher. He was said to be a remarkably bold and dexterous operator. In 1900 he went to Chicago, became Professor of Clinical Surgery in the College of Physicians and Surgeons, and subsequently, Professor of Surgery at the Chicago Post-Graduate Medical School and Hospital; Surgeon-in-Chief to the Chicago Hospital; and Surgeon to the Cook County Hospital for the Insane. His departure from Canada had been referred to as a public calamity. The hospitals which he left in Winnipeg refused to accept his resignation and maintained his name on the rolls of their staffs up to the time of his death. He was a forthright, vigorous man, intolerant of fraud or deception, and given to couching his outspoken remarks in a trenchant and vigorous style. One obituary characterizes him as strong, rugged, ardent, enthusiastic, positive, and aggressive.

In Winnipeg he had gained much of his reputation as a result of his boldness in dealing with hydatid disease of the liver, then common in the drivers of dog teams, and had there begun to develop and become known for his operations for inguinal hernia and cleft palate. *The Technique of Modern Operations for Hernia,* published in 1908, was at once hailed as a classic. He wrote also on gall bladder disease, thoracoplasty, cleft palate, and on a number of facets of urologic and gynecologic disease. He died of "general septicemia and other complications resulting from a carbuncle."

A. H. Ferguson of Chicago in 1899 wrote a hard-hitting, no quarter asked—or granted—paper entitled "Oblique Inguinal Hernia, Typic Operation for Its Radical Cure," which was published in *The Journal of the American Medical Association* that year. Ferguson introduced the description of his operation by stating, "In response to a firm conviction of having found an improvement on the older operations, independent thinking surgeons invented the different procedures we now possess." He went on to say, "It is plainly observable that 'Do thus and he doeth it' without giving the reasons why, has been too frequently


A. H. FERGUSON
1853–1911

followed by the profession. It is now nearly five years since I undertook to criticize the operations then in vogue, for I was not wholly satisfied with any of the methods.'' He remarked that the ''combination'' method he evolved was followed by some recurrences at the upper angle, so that he was led to the resolution that deficient attachment of the internal oblique and transversalis to Poupart's ligament is the probable cause of recurrence.

He described the classic herniorrhaphy without transplantation 4 years before Halsted published a description of it, although Halsted had undoubtedly been employing such a procedure for some time. [Actually, a review of the records of The Johns Hopkins Hospital confirms Halsted's statement that he had been experimenting from the first with the operation in which the cord was left undisturbed. Surgical Cases Nos. 448 and 460, in 1890, were operated upon in this way, without transplantation of the cord.]

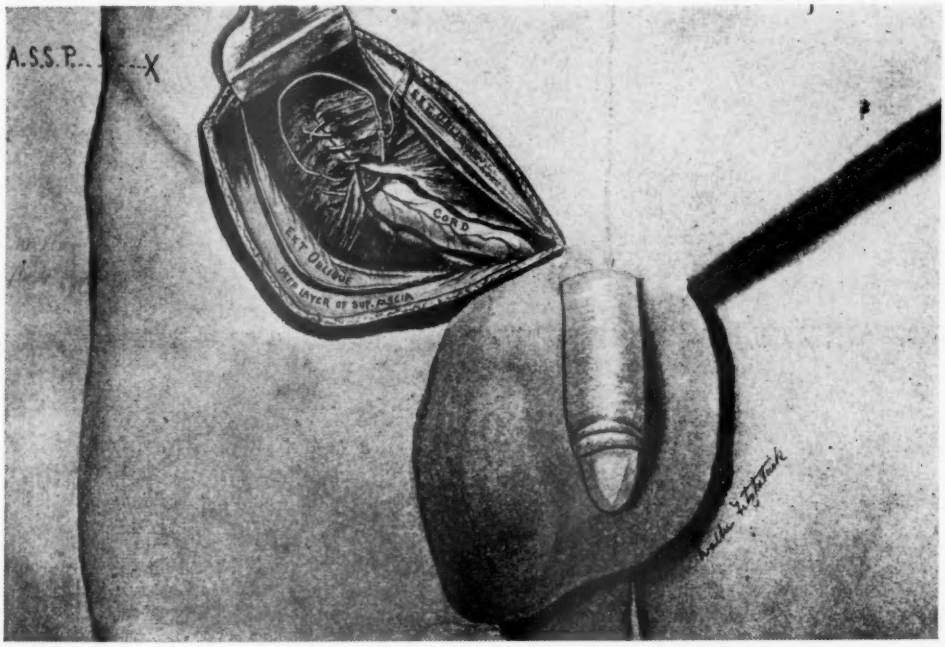


Fig. 11. The semilunar skin incision has been made arching from a point over Poupart's ligament 1½ inches below the anterior-superior spine, upward and medially, turning downward to the pubic bone. The aponeurosis of the external oblique has been stripped, cleaned, and split, so as to divide the external ring. The flaps have been dissected back and the sac exposed, opened, ligated high, amputated, and allowed to retract. Ferguson laid strong emphasis on the inadvisability of disturbing the position of the cord in any way. The fibers of the transversalis fascia are being sutured to tighten up the internal ring. Ferguson generally ignored the cremaster unless it was bulky, in which case he excised it.

1. The Ferguson skin incision is semilunar, beginning 1½ inches below the anterior superior spine of the ilium, and extending medially and then downward, terminating over the conjoined tendon, near the pubic bone.

2. The external abdominal ring is cut through and the longitudinal fibers of the aponeurosis of the external oblique separated directly over the inguinal canal "far beyond the internal ring over the surface of the internal abdominal oblique muscle and up under the skin to a point nearly opposite the anterior superior spine of the ilium." (p. 7.) The flaps of the aponeurosis are retracted. Ferguson considered "the congenital deficient origin of the internal oblique and transversalis muscles one of the most frequent and important causes of oblique inguinal hernia. When the structures are well defined and not too much weakened by pressure atrophy a typic operation can be proceeded with."

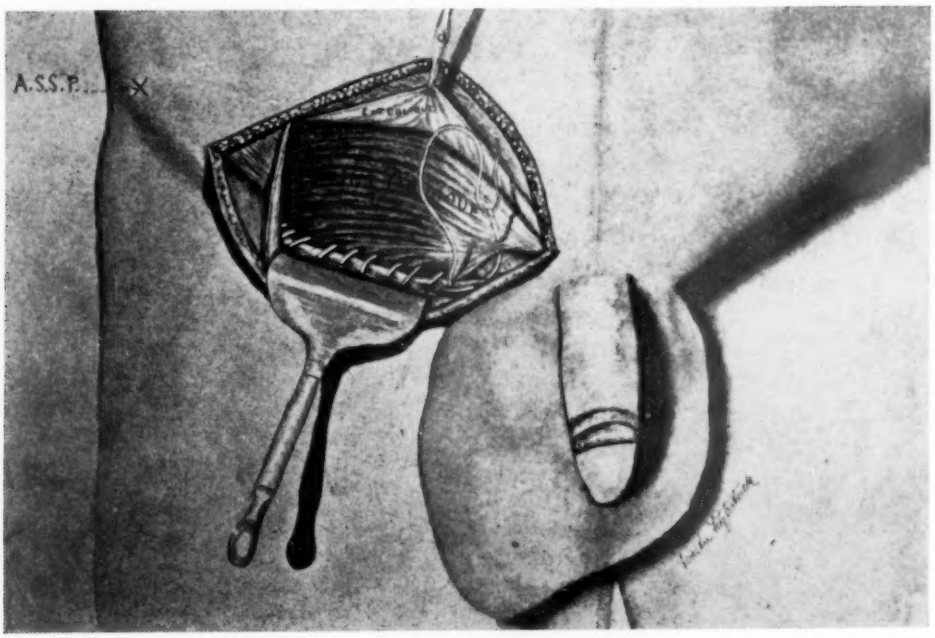


Fig. 12. "The internal oblique and transversalis muscles suture to the internal aspect of Poupart's ligament, and restore to their normal origin. I usually . . . extend the sewing fully two thirds down Poupart's ligament. . . . It is surprising how easily these two structures come together without the least discernible tension, and it is gratifying to observe how perfectly these powerful muscles cover and protect the internal abdominal ring and inguinal canal."

3. The sac is dissected from the cord, opened, and ligated high, cut off, and the stump dropped back.

4. "The cord is not disturbed, I have never been satisfied with the raising and transplantation of the cord. In more cases than have been recorded the testicle has come to grief by this unnecessary procedure. Tearing the cord out of its bed is without an anatomic reason to recommend it, a physiologic act to suggest it, an etiological factor in hernia, congenital or acquired to indicate it, nor brilliant surgical results to justify its continuance. Leave the cord alone, for it is the sacred highway along which travel vital elements indispensable to the perpetuity of

our race. The veins in the cord are not disturbed, unless a varicocele complicates the hernia. If the cremasteric fibers are unduly thickening the cord they would better be removed along with any adventitious tissue that is not infrequently present." (p. 9.)

(Halsted's 1903 paper, 4 years later, indicated that he had from the first experimented with nontransplantation of the cord and now regularly left the cord undisturbed. The date when this became regular practice is not given.)

5. The internal ring is reconstructed by interrupted or continuous sutures in the transversalis fascia lateral to the cord.

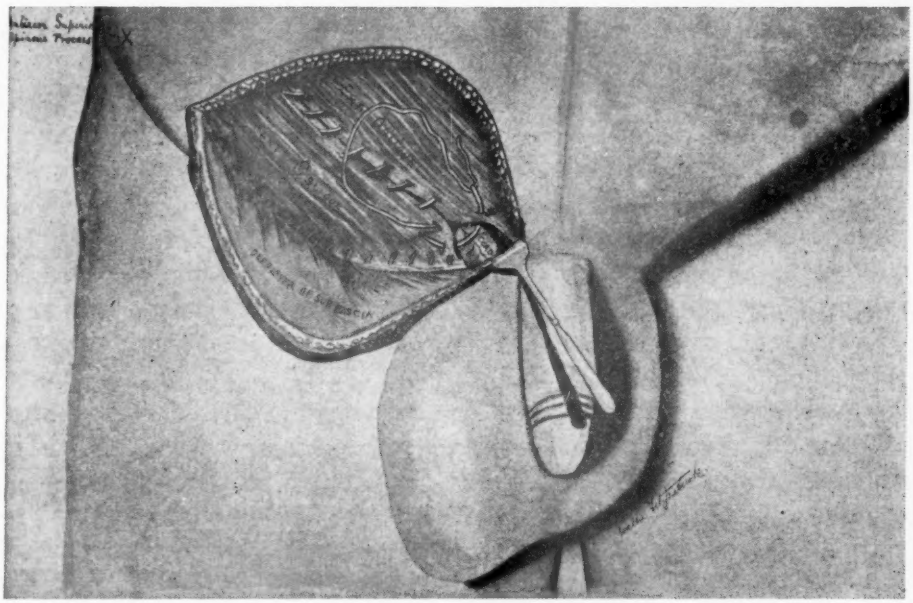


Fig. 13. "Bring together the separated edges of the aponeurosis of the external oblique muscle, restore the external abdominal ring."

6. "The internal abdominal oblique and transversalis muscles are sutured to the internal aspect of Poupart's ligament and restored to their normal origin. . . . Extend the sewing fully two thirds down Poupart's ligament. It is surprising how easily these two structures come together without the least discernible tension, and it is gratifying to observe how perfectly these powerful muscles cover and protect the internal abdominal ring and inguinal canal." (p. 10.)

7. The separated edges of the aponeurosis of the external oblique muscle are brought together and the external abdominal ring restored.

"*Commendable Features.*

"The different structures in the abdominal wall are placed in their normal relationship.

"A. The tying of the sac restores the normal rotundity of the peritoneum.

"B. The suturing of the transversalis fascia, forming a new internal ring at the same time, obliterates the hernial infundibuliform process.

"C. Sewing the internal oblique and transversalis muscles to Poupart's ligament secures a normal origin for them and they then find [sic] perfect protection to the internal ring, cord, and canal.

"D. The suturing of the separated fibers to the aponeurosis of the external oblique protects the underlying muscles and cord, while the skin flap covers all."

As for his results, Ferguson stated that in the previous 18 months he had performed this operation 64 times, with as yet no relapses. The patients varied in age from 5 to 76 years. There was one death, that of an old man of 74 who died 4 days later from "the effect of the anesthetic." There were 3 instances of suppuration. There were 4 strangulated hernias, all of which healed cleanly. Rubber gloves were used in only 6 operations on 3 patients. The rest of these operations were performed with bare hands but the fingers were allowed to touch the tissues "as little as possible." "In about half the cases chromic-formed catgut No. 0- 1- 2- and 3 was used in skin, as well as the deeper structures. In the last six months I have discarded No. 2 and 3." "The patients are kept in bed from 21 to 28 days, enjoined not to assume any work for six weeks after operation, and advised to wear a broad support—no truss—for three or four months."

In the discussion Ferguson laid further great stress on the fact that, in patients with inguinal hernia, the internal oblique muscle has a deficiency in its most medial attachment to Poupart's ligament. There is an accompanying section indicating from a large number of human dissections that the internal oblique ordinarily is attached to "rather more than the outer two-thirds of ligament."

Ferguson finishes by discussing "atypic operations." (p. 14.) "Atypic operations for the radical cure of oblique inguinal hernia are all those which do not, in the completed operation, leave all structures as they are present in a normal region. It is not necessary to mention these, but I wish to draw attention to plastic procedures on the sheaths of the rectus muscle, when the conjoined tendon is abnormally thinned out (Bloodgood), to the utilization of the sac (McEwen), and to the employment of the sartorius muscle to strengthen the inguinal region (Ferguson).

"Some cases may present themselves—but they must be comparatively rare—where it may be advisable to do plastic work in addition to the typic operation."

It is interesting that Halsted and Bassini are not mentioned anywhere in the paper, except by implication in the opening paragraphs which demolish all methods of hernia repair other than Ferguson's.

REFERENCE

Ferguson, A. H.: Oblique Inguinal Hernia. Typic Operation for Its Radical Cure, J. A. M. A. 33: 6-14, 1899.

Editorial

CONDUCTED BY I. S. RAVDIN, M.D., AND J. GARROTT ALLEN, M.D.

A Plea for Complete Removal of the Antral Segment in the Performance of Vagotomy and Distal Gastrectomy for Duodenal Ulcer

Approximately 13 years ago, Smithwick first performed vagotomy and an estimated 50 per cent distal gastrectomy for the treatment of the complications of duodenal ulcer. At nearly the same time Edwards independently employed vagotomy with a conservative resection for the treatment of the disease. He termed the distal resection an "antrectomy," which encompassed removal of an estimated 40 per cent of the stomach. Smithwick and Edwards felt that these procedures not only constituted a physiologic surgical approach to the problem of duodenal ulcer aimed at eliminating the 2 main sources responsible for gastric secretion, but also possessed the added advantage of preserving a generous portion of the gastric reservoir. Postoperative gastric secretory studies following each procedure revealed a complete achlorhydria during the fasting state and following stimulation with a test meal. Over 90 per cent of the patients tested had no free acid response when challenged with histamine. The 12-hour quantitative level of gastric juice was reduced to an exceedingly low figure.

In order for vagotomy and conservative gastrectomy to prove effective in preventing marginal ulceration, it has been repeatedly stressed that the *entire* gastric antrum must be excised in combination with a complete vagotomy. Unfortunately, the gastric antrum has no precise gross anatomic delineation, and to be assured of its complete removal a distal resection comprising an estimated 40 to 50 per cent of the entire stomach should be performed. The resection is usually carried to a higher level on the lesser curvature than on the corresponding greater curvature, as the antral glands may on occasion extend 1 to 2 cm. proximal to the incisura angularis. Smithwick[2] has shown by carefully performed secretory studies that when vagotomy is carried out in combination with a distal resection of lesser degree, as for example an estimated 20 to 30 per cent distal gastrectomy, significant peptic activity continues to persist in the residual gastric pouch. In the early 1940's, Edwards[1] performed several operations consisting of vagotomy with an estimated 20 to 30 per cent distal gastrectomy, and subsequent secretory studies showed that with test meal and histamine stimulation significant peptic activity was still present. The pH of the gastric juice under these circumstances remained within the range of that seen in patients with active ulcer disease. The postoperative gastric

secretory values obtained closely paralleled those in patients who had been subjected to vagotomy with a drainage operation. It is now well appreciated that this latter operation is usually associated with a recurrent ulcer rate varying from 5 to 10 per cent.

Recently a few reports have appeared in the surgical literature advocating vagotomy in combination with an estimated 25 per cent distal resection for the surgical treatment of duodenal ulcer, the amount of tissue removed being termed an "antrectomy." One report has stated that the resection represents only one-fourth, or even less, of the entire stomach. Although a resection of this limited scope may at times include the complete antral segment, it is feared that in many instances antral tissue will be retained proximal to the line of resection. The short follow-up of reported patients who have undergone this limited resection with vagotomy is of no value at present in determining recurrence rates. However, from our own experience and from that of others, it is reasonable to assume that the procedure would not afford the degree of protection against ulceration which is provided by the operations developed by Smithwick and Edwards.

When vagotomy is combined with a limited distal resection of the Billroth II type, retained antral tissue proximal to the line of resection is bathed by the alkaline regurgitant contents of the jejunum, which serve as a stimulus for the release of gastrin by the antral mucosa. Distal antral exclusion following vagotomy and a sleeve resection is also associated with persistent acid stimulation, for an already high rate of recurrent ulceration has been described by its original advocates.

Therefore, a plea is made for the removal of the gastric antrum in its entirety in the performance of vagotomy and distal gastrectomy for duodenal ulcer.

REFERENCES

1. Edwards, L. W., and Herrington, J. Lynwood, Jr.: Vagotomy and Gastroenterostomy—Vagotomy and Conservative Antrectomy, Ann. Surg. **137**: 873, 1953.
2. Farmer, D. A., Howe, C. W., Porell, W. J., and Smithwick, R. H.: The Effect of Various Surgical Procedures Upon the Acidity of the Gastric Contents of Ulcer Patients, Ann. Surg. **134**: 3, 1951.

J. Lynwood Herrington, Jr., M.D.
2001 Hayes Street
Nashville, Tennessee

Erratum

In the Editorial, "Selecting the Operation for the Particular Patient in Cases of Duodenal Ulcer," by J. Lynwood Herrington, Jr., in the March, 1960, issue of Surgery, in Table I, line 1, the series of Edwards and Herrington, the number of recurrences should be 4 instead of 15 and the total of that column should be 12.

In the third line of the third paragraph, the number 13 should also be 12.